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AN ASSESSMENT OF BURIED SILK SUTURES IN LENS SURGERY

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The first recorded lens extraction was performed by the French ophthalmologist, Daviel, in 1745 (quoted by Stallard[3]). Since that time surgeons have continually striven to make this procedure safer. One of the most significant advances in this respect has been the use of sutures to close the limbal wound.

Williams, in 1867,[4] was the first to use a corneal suture. Little interest was shown in this idea for many years, for two main reasons. These were: the lack of the very fine needles and silk required, and the reluctance to prolong an operation which was already full of hazards on account of the lack of orbicularis and retrobulbar akinesia. It was only in the 1930s that Stallard,[3] Lindner,[5] Verhoeff[6] and Maclean[7] revived interest in corneal suture after lens extraction. The main advantage of this is the security afforded by adequate closure of the limbal section which, in the intracapsular method, occupies 180° in the upper half of the limbus. In the extracapsular method it is rather smaller. On the other hand these sutures have to be apposed with the greatest accuracy otherwise serious complications can result, e.g. a flat anterior chamber, secondary glaucoma, delayed wound healing, anterior chamber fistula, epithelial downgrowth, and high astigmatic correction. One to nine interrupted corneo-scleral sutures are used, depending on the individual surgeon.

There are a large number of technical variations but, broadly speaking, 'exposed' sutures fall into 2 major groups:

1. *Pre-placed corneo-scleral sutures.* The suture is placed in a groove cut into the limbal sclera for half its thickness, at the 12 o'clock meridian, before making the cataract section. The 2 arms of the suture are pulled out of the groove and separated, and the section is then made between them. In this way the wound edges are very accurately apposed when the suture is tied.

2. *Post-placed corneo-scleral suture.* The section is first cut and then the suture placed. A limbal-based conjunctival flap may be fashioned if so desired. The sutures are inserted, first through the corneal side, the needle passing through half the corneal thickness, and then through the scleral side, taking great care to achieve accurate apposition.

These 'exposed' sutures have 2 great disadvantages: (*a*) they irritate the eye postoperatively; and (*b*) they have to be removed after 10 days, a potentially dangerous procedure which has spoilt a good operation in many cases.

Recently Barraquer[1] introduced a technique using very fine silk sutures (Belgian virgin silk) which are post-placed and tied under the conjunctival flap and left there. They are completely non-irritant, and are not removed. Over a period of a few months they work their way out silently through the conjunctiva. Because of these advantages, I believe this technique should replace existing methods. The results, when using these sutures, are as good as with 'exposed' sutures. An analysis of the results of 75 unselected cases, treated by this method from November 1957 to November 1958, is given below and, in my opinion, proves its superiority to the older forms of treatment. All these operations were performed by me at St. Mary's Hospital, Paddington, London; as a control the results, using more classical techniques at Groote Schuur Hospital,[2] Cape Town, are quoted.

MATERIAL AND METHOD

The cases were selected at random so that they included senile, traumatic, secondary, complicated and uni-ocular cataracts. A minimum 3-month period between operation and follow-up was allowed in all cases.

In the St. Mary's Hospital series of 75 cases, 65 had accurate refractions recorded at the time of the follow-up. These refractions and assessments of postoperative visual acuity were all done at a refraction clinic by independent observers, and not by myself.

In the Groote Schuur Hospital series, there were 100 cases taken at random from the European ward only, to rule out any possible racial differences. Forceps were used in 85 cases, an erisophaeke in 13 and a vectus in 2. Refractions and assessments of postoperative visual acuity were done in the out-patient clinic. Sutures were used on all cases but no buried sutures were used. The operations were performed by 12 surgeons, 2 of whom were on the resident staff.

TECHNIQUE USING BURIED SUTURES

The same technique was used in placing the buried sutures in all cases. It was decided that, theoretically, 3 well-placed sutures should be sufficient to close the limbal wound adequately, and this number was employed in all cases.

Anaesthetic

Local and general anaesthesia was used at random. In one case where local anaesthesia was used, vitreous was

lost due to the restlessness of the patient. There were no complications when general anaesthesia was used, and this was by far the pleasanter method both for the surgeon and the patient.

Type of Extraction

In all but one of the cases the section and flap was cut with a Graefe knife in the upper half of the limbus. In one case the section was made in the lower half, since an operation for iris inclusion had been done previously. Three post-placed virgin silk sutures were inserted at the 10 o'clock, 12 o'clock and 2 o'clock positions. A peripheral iridectomy was then done in all but one case — here tough posterior synechiae demanded a broad iridectomy. Capsule forceps were used to extract the lens in all except 6 cases (8%) where a motor-controlled erisophaeke was used. No difference was noticed in the results. Alpha-chymo-trypsin was used in only 1 case with a good result.

TABLE I. TYPE OF CATARACT EXTRACTION

Type of extraction	*St. Mary's**	*Groote Schuur***
Intracapsular	80%	63%
Extracapsular	20%	37%

* St. Mary's Hospital series.
** Groote Schuur Hospital series.

An intracapsular extraction was attempted in all cases; this failed in 15, i.e. in 20% (Table I).

When the lens had been removed the sutures were tied and the conjunctival flap was stroked into place over them.

Postoperative Management

Double pads were placed over the patients' eyes and all patients were put to bed. The first dressing was done by the ward sister after 24 hours, at which time 1 eye was uncovered and the patients were allowed to get up. In intracapsular extractions pilocarpine drops were used on the operating table and for 3 days postoperatively. In failed intracapsular extractions atropine drops were used initially.

RESULTS

1. *Visual Results*

For the sake of comparison these were assessed on an arbitrary scale: 6/6 or better was rated as 1, 6/9 as 2 and so on up to 6/60 as 7. Ability to count fingers was 8, seeing hand movements was 9, perception of light was 10, and a blind or lost eye was 11.

The average visual result at St. Mary's Hospital was 2·2 (about 6/9) and at Groote Schuur Hospital it was 3·1 (about 6/12).

TABLE II. COMPLICATIONS IN THE 2 SERIES OF OPERATIONS

Complications	*Incidence*	
	*St. Mary's**	*Groote Schuur***
None	89·6%	57%
Hyphaema	2·6%	29%
Vitreous haemorrhage	Nil	2%
Vitreous loss	2·6%	2%
Choroidal detachment	2·6%	2%
Anterior chamber leak	1·3%	Nil
Retinal detachment	Nil	3%
Retinal and choroidal detachment ..	Nil	1%
Prolapsed or incarcerated iris. ..	1·3%	4%
Average astigmatism	+ 2·0 D	+ 1·87 D

* St. Mary's Hospital series.
** Groote Schuur Hospital series.

2. *Analysis of Complications*

Complications in the 2 series are compared in Table II.

In the St. Mary's Hospital series 89·6% of cases were entirely free of complications and in the Groote Schuur Hospital series, 57%. This difference is attributable to the high incidence of hyphaema in the Groote Schuur Hospital series. This generally resolves without serious sequelae.

DISCUSSION

The results in both series are comparable. There are no statistically significant differences apart from the much lower incidence of hyphaema, prolapsed iris and retinal detachment in the St. Mary's Hospital series. As the incidence of hyphaema is related to wound healing, this may be connected with the type of suture used. However, so many other factors are involved, e.g. the use of a conjunctival flap, the use of a keratome and scissors or Graefe knife, that it is impossible to be dogmatic about this.

The advantages of using buried silk sutures which are safer and therefore more desirable, are:

1. The sutures are less irritating postoperatively, and also less irritating than buried catgut sutures.

2. The sutures do not require removal, which can be a trying experience for the patient and may lead to a mishap.

3. The sutures can be used with a conjunctival flap which reduces the possibility of epithelial downgrowth.

Experience with this method has also brought to light its disadvantages. These are essentially those of a post-placed suture whatever its type. Where the anterior chamber is filled with vitreous humour pre-operatively, e.g. following trauma, use of a pre-placed suture does provide better control. In these cases, which are always difficult, each surgeon must use his discretion and the technique which suits him best.

SUMMARY

The advantages of using post-placed buried sutures are stressed. By comparing a series where these are used with a control series using 'exposed' sutures, it is shown that the results of this method are comparable with those where 'exposed' sutures are used.

My thanks are due to the surgeons at the Western Ophthalmic Hospital (St. Mary's Hospital, London), for entrusting me with the treatment of their cases and for allowing me to use their case records for this analysis.

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EDITORIAL : VAN DIE REDAKSIE

NEGOTIATIONS WITH MEDICAL AID SOCIETIES AND INSURANCE COMPANIES

The Medical Association has recently passed through an extremely difficult period in its negotiations with the insurance companies which undertake medical insurance. Much criticism has been levelled at the actions of the Association — fortunately it was almost always criticism of a more or less positive nature. The representatives of the Association who had the difficult task of carrying out the negotiations used this constructive criticism as a guide to the feelings of members concerning this problem. Therefore, members can rest assured that their representatives in these negotiations acted at all times in the interests both of the doctors and of the patients who are entrusted to their care.

To gain a proper perspective of the whole problem, it is necessary to go back a few years to the beginnings of the medical aid society movement in this country. With the rise in the cost of living many people in the lower income groups found it difficult to meet their doctors' bills. Medical aid societies were formed as a solution to this problem. These societies had to comply with certain requirements before recognition by the Association, and they had to agree to pay doctors direct and in full — in turn, they were allowed a preferential tariff of fees. This agreement was reached after prolonged negotiations. Thus the medical profession, through the Association, obtained something tangible — their accounts were paid direct and in full and the problem of bad debts was minimized for many doctors. They were also able to treat many patients in their surgeries, whom they would previously have had to treat at hospitals or clinics. On the other hand, doctors performed a valuable service as part of their commitments under the medical aid scheme — they provided private attention, at medical aid tariff rates, for many patients who could not otherwise have afforded this private attention. In this way the agreement with the medical aid societies was an important and constructive step forward in the patient-doctor relationship in South Africa.

This system worked extremely well for all concerned for many years. Meanwhile, the cost of living, and especially the cost of medical services and medicine, rose higher and higher. At this stage the insurance companies entered the field of pre-paid medical care.

At first the two large insurance companies which undertook the provision of this service, decided on their own to operate on a tariff of fees which was higher than that of the preferential tariff, *viz.* the preferential tariff plus 25 and 33⅓ per cent. respectively. The premiums charged were obviously too low to cover this tariff, and eventually both insurance companies began to pay their members' accounts on the basis of the preferential tariff which had been agreed upon between the Medical Association and the medical aid societies, although there was no official recognition of these insurance companies by the Association. Furthermore, their insured members did not all fulfil the conditions laid down for medical aid society members. In other words, the insurance companies fixed their tariffs at medical aid rates even for those persons in the higher income groups who could well afford private fees.

This principle of payment was naturally condemned most strongly by the Medical Association. Matters were made worse when one of the insurance companies began to issue 'coded' cheques on behalf of its members on the basis of the preferential tariff of fees, and expected doctors to accept these cheques 'in full settlement' of their accounts. This state of affairs was quite unacceptable and caused great concern and dissatisfaction among members of the Association.

Now began a long series of difficult negotiations. For the Association the position was fraught with dilemmas, since its members were in many ways divided in their attitude to the problem. For instance, some members accepted the 'coded' cheques, while others returned them to the insurance company as a matter of principle.

It was difficult, if not impossible, for the Medical Association to find a basis for negotiation with the insurance companies which would be acceptable to them and which would satisfy *all* the members of the Association at the same time. The Association therefore followed the only path which was possible in this difficult period — its representatives continued to negotiate as far as they were able, trying not to do anything which would bring the dignity of the Association and the profession into disrepute.

At its recent meeting in Vereeniging, the Federal Council officially recognized as medical aid societies a large number of groups administered by the two insurance companies. It must be remembered that all these groups satisfied the criteria for this recognition, in the same way as the older long-approved medical aid societies did. As a basis for further negotiations, it appeared that the insurance companies preferred an income ceiling for their members below which the preferential tariff would operate. The existing medical aid societies, on the other hand, preferred to continue to operate, as they have always done, on an average income figure for their members.

The Association, through the Executive Committee of Federal Council, decided to accept a ceiling of £2,300 as a basis for negotiation. The Association is aware that many members do not agree with this figure, but some position had to be reached where negotiations could be conducted. The insurance companies had suggested a still higher figure, the Association a lower one. When this point was reached, the negotiations with the South African Mutual Medical Aid Society broke down completely. One of the main reasons for this break was that this insurance company would not agree to withdraw the 'coded' cheques.

To sum up, the position at the moment is as follows:

1. The approved medical aid societies, which function on their own and are not underwritten by the insurance companies, remain as they were and are still recognized as in the past. Payments for their members are made

according to the preferential tariff of fees. A list of these societies was published on page 1062 of the issue of the *Journal* for 10 December 1960.

2. A large number of groups which comply in all respects with the requirements for recognition as medical aid societies, but which are administered by SANSOM, were recognized at the last meeting of Federal Council. A list of these groups was published as a supplement to the *Journal* of 26 November 1960. Since these groups operate as medical aid societies, doctors will be paid for their services to members of these groups direct and in full by SANSOM at the preferential tariff of fees.

3. The level of £2,300 has been accepted in principle as a basis for recognition of other groups which are administered by SANSOM.

4. No recognition is given to *any* group insured by the South African Mutual Medical Aid Society. Those groups administered by this company, which had been approved by Federal Council at its meeting in October 1960 as fulfilling the requirements for medical aid societies (published on page 1064 of the *Journal* for 10 December 1960) also fall away as recognized groups. We have briefly given the reasons for this withdrawal of recognition from all persons insured by the South African Mutual Medical Aid Society; they were set out as well in the Official Announcement and the column 'From the Secretary's Desk' on pages 35 and 36 of the issue of the *Journal* for 14 January 1961.

5. The Medical Services Plan, which is sponsored by the Association, is at present in operation on the Witwatersrand as a pilot scheme.

It would facilitate matters for the negotiating committee if members would not make personal unilateral arrangements with the insurance companies or their agents. They should rather bring their difficulties to the attention of the Head Office of the Association, or discuss them at meetings of Branches.

Having outlined the present position, we wish to make an urgent plea to all members of the Medical Association to support the Association in this difficult matter. There may still be uncertainty for some time in the field of medical insurance, but progress, in the interests of our members, will continue. Only if we all stand together can we maintain the integrity and dignity of the profession in the face of the demands of other organizations. Furthermore, it is only by standing together that we will be able to ensure that the interests both of our patients and of ourselves are safeguarded, and that no inroads are made into our relationship with our patients or the high standing and honour of our profession.

ONDERHANDELINGE INSAKE MEDIESE HULPVERENIGINGS EN VERSEKERINGSMAATSKAPPYE

Die Mediese Vereniging het 'n moeilike tydperk van onderhandeling met die versekeringsmaatskappye agter die rug. Daar was baie kritiek op die optrede van die Vereniging — gelukkig was dit egter altyd van 'n min of meer positiewe aard. Die verteenwoordigers van die Vereniging wat met die moeilike taak van onderhandeling belas was, het die konstruktiewe kritiek wat daar was gebruik om vir hulle as leidraad te dien; daarom kan die lede van die Vereniging verseker wees daarvan dat die optrede van diegene wat namens die Vereniging onderhandel het, in die belang van die lede van die Mediese Vereniging is, sowel as in die belang van die pasiënte wat aan hulle sorg toevertrou is.

Om 'n perspektief op die saak te kry, sal dit goed wees om effens terug te kyk. Om mee te begin, het daar 'n aantal jare gelede die stelsel van mediese hulpverenigings ontstaan. Aangesien die lewenskoste só gestyg het dat baie persone in die laere inkomstegroepe dit moeilik gevind het om hul doktersrekeninge te betaal, is besluit om dié persone tegemoet te kom. Dit het dan gelei tot die stigting van die nou reeds bekende mediese hulpverenigings. Hierdie verenigings moes almal aan sekere vereistes voldoen en hulle moes die rekeninge van dokters direk betaal — in ruil waarvoor 'n voorkeurtarief aan hulle toegestaan is. Die dokters het dus iets tasbaars uit die transaksie gekry — hulle rekeninge is direk betaal en dit het die probleem van slegte skulde vir baie dokters verlig. Ook kon hulle nou 'n groot deel van die pasiënte teen betaling behandel, wat hulle anders tog maar vry sou moes behandel in hospitale en klinieke. Aan die ander kant het die dokters egter ook iets gegee. Deur die voorkeurtarief toe te staan, het hulle die dienste wat hulle as private geneeshere kan lewer, na 'n groter groep mense uitgebrei teen relatiewe verminderde besoldiging. Alhoewel die dokters dus voordeel uit die ooreenkoms getrek het, het hulle ook 'n vergrote *diens* gelewer. Dit is die basis van die ooreenkoms met die hulpverenigings. Hierdie ooreenkoms, insluitende natuurlik die vasstelling van die voorkeurtarief, het eers na langdurige onderhandeling tot stand gekom.

Hierdie stelsel het oor jare redelik goed beantwoord. Daarna het die toestand verander deurdat die groot versekeringsmaatskappye ook toegetree het tot die veld van mediese versekering.

Aan die begin het die twee groot versekeringsmaatskappye onderneem om meer uit te betaal as die voorkeurtarief, naamlik, voorkeurtarief plus 25 en 33⅓% respektiewelik. Die premies het egter te laag geblyk te wees, en altwee maatskappye het begin om op die basis van die voorkeurtarief uit te betaal, sonder egter dat die groepe van hul versekerde lede aan die vereistes van die hulpverenigings voldoen het. Met ander woorde, die maatskappye het uitbetaal teen die voorkeurtarief ook vir persone wat genoeg verdien het om as private pasiënte te geld.

Hierdie beginsel is natuurlik baie sterk afgekeur deur die Mediese Vereniging. En sake is vererger toe een van die maatskappye begin het om tjeks ten opsigte van hul versekerde lede aan te bied op die basis van die voorkeurtarief terwyl hulle van die dokters verwag het om die tjeks aan te neem ‚in volle vereffening' van hul rekening. Dié toestand van sake het groot ontevredenheid veroorsaak by lede van die Mediese Vereniging en kon nie aanvaar word nie.

'n Lang reeks van moeilike onderhandelinge het nou gevolg. Vir die Mediese Vereniging was die toestand vol dilemmas, aangesien sy eie lede so verskil het oor die saak.

Baie lede het byvoorbeeld die genoemde tjeks aanvaar, terwyl ander lede die tjeks op beginsel nie wou aanvaar nie. Dit was moeilik, feitlik onmoontlik, vir die Mediese Vereniging om met die versekeringsmaatskappye te onderhandel op 'n basis wat hulle sou aanvaar en wat *al* die lede van die Vereniging terselfdertyd sou tevrede stel.

Die Mediese Vereniging het dus die enigste weg gevolg wat vir hom moontlik was — hy het naamlik deur sy verteenwoordigers bly onderhandel volgens die lig wat hy gehad het, en altyd met die oog daarop om amptelik niks te doen wat die waardigheid van die Vereniging en die professie sou skaad nie.

By sy laaste vergadering op Vereeniging, het die Federale Raad 'n groot aantal groepe wat deur die twee versekeringsmaatskappye geadministreer word, as hulpverenigings goedgekeur. Dit moet onthou word dat al hierdie groepe voldoen het aan die vereistes van goedgekeurde mediese hulpverenigings. Dit het egter geblyk dat dit met die oog op toekomstige onderhandelinge geriefliker sou wees vir die versekeringsmaatskappye om te voldoen aan 'n inkomsteperk vir hul lede op wie die voorkeurtarief van toepassing sou wees. Die bestaande mediese hulpverenigings sou nog voldoen (soos voorheen) aan 'n gemiddelde inkomstesyfer van hul lede.

Die Vereniging het deur die Uitvoerende Komitee van sy Federale Raad besluit om 'n perk van £2,300 te aanvaar as basis van onderhandeling. Die Vereniging is bewus dat baie lede nie hiermee sal saamstem nie, maar êrens moes tog 'n besluit geneem word op grond waarvan verdere onderhandelinge gevoer kan word. Op hierdie stadium het die onderhandelinge met die Suid-Afrikaanse Onderlinge Mediese Hulpvereniging egter totaal afgebreek, aangesien hierdie hulpvereniging onder andere nie die gekodifieerde tjek nou wil terugtrek nie.

Die toestand van sake staan dus, om op te som, op die oomblik soos volg:

1. Die goedgekeurde mediese hulpverenigings wat op hul eie funksioneer en wat nie deur versekeringsmaatskappye onderskryf word nie, bestaan en word nog erken soos voorheen. Uitbetalings vir dienste aan hul lede word, soos in die verlede, gemaak op die basis van die voorkeurtarief. 'n Lys van hierdie hulpverenigings is gepubliseer op bladsy 1062 van die uitgawe van die *Tydskrif* van 10 Desember 1960.

2. 'n Groot aantal groepe wat in alle opsigte voldoen aan die vereistes van mediese hulpverenigings, maar wat deur SANSOM geadministreer word, is goedgekeur by die laaste vergadering van die Federale Raad. 'n Lys van hierdie groepe is as Byvoegsel tot die *Tydskrif* gepubliseer op 26 November 1960. Aangesien hierdie groepe voldoen aan die vereistes van goedgekeurde mediese hulpverenigings, sal dokters op die basis van die voorkeurtarief vir hulle direk en ten volle uitbetaal word.

3. In beginsel is die perk van £2,300 aanvaar vir die goedkeuring van *verdere* groepe wat deur SANSOM geadministreer word.

4. Geen goedkeuring word meer verleen aan enige groep wat deur die Suid-Afrikaanse Onderlinge Mediese Hulpvereniging verseker is nie, aangesien alle onderhandelinge nou met hierdie hulpvereniging beëindig is, soos ons aangetoon het en soos ook verduidelik is in die amptelike verklarings en die rubriek van die Sekretaris wat op 14 Januarie 1961 op pp. 35 en 36 van die *Tydskrif* verskyn het. Die groepe wat deur hierdie vereniging geadministreer word en wat voorheen goedgekeur was as mediese hulpverenigings (p. 1064, *Tydskrif* van 10 Desember 1960), verval nou ook as goedgekeurde groepe.

5. Die Mediese Diensplan, wat die ondersteuning van die Vereniging geniet, is op die oomblik in werking op die Witwatersrand, as eksperiment.

Dit sal sake vir die onderhandelende Komitee baie vergemaklik as lede van die Vereniging nie persoonlike ooreenkomste met die maatskappye of hul agente aangaan nie maar liewer hul moeilikhede by hul takvergaderings bespreek en/of hulle na die Hoofkantoor van die Vereniging verwys.

Langs hierdie weg wil ons by herhaling 'n dringende beroep op alle lede van die Mediese Vereniging doen om die Vereniging in sy moeilike taak te ondersteun. Daar sal miskien nog vir 'n tyd lank onsekerheid heers, maar vordering in belang van die lede word nog steeds gemaak. Slegs deur almal saam te staan kan ons verhoed dat ons as professie oorheers word deur die belange van groot sakeondernemings. En slegs deur saam te staan kan ons reg laat geskied aan die behoeftes van ons pasiënte en aan onsself — en ook waak daarteen dat die waardigheid van die professie ondermyn word.

TAALRUBRIEK

Die Taalkomitee van die Geneeskundige Skool van die Universiteit van Stellenbosch stel voor om te gebruik:

1. Eng. *boost, booster:* Afr. *skraag, skrager*. Daarnaas kry ons dan bv. skraagmiddel, skraagdosis, skraagtempo (Eng. booster rate) e.d.m. Die Komitee het die woorde versterk, versterker, versterking ook ernstig oorweeg, maar die groot beswaar teen hulle is dat hulle betekenisse so vaag en algemeen is, dat hulle dus in verskeie soorte sinsverband gebruik kan word. *Skraag* is nie baie bekend nie, miskien nog die beste in die woord *onderskraag*, daarom is hy dienliker vir iets afgebakends soos die Eng. boost.

2. Eng. *focus:* Afr. *fokus*, as s.nw. *èn as ww*. Dit is moontlik dat *fokus* deur sommige mense onder Engelse invloed as werkwoord gebruik word, maar bewys kan ons dit nie. Daarby kom nog dat Afr. honderde s.nwe. sonder meer as wwe. gebruik — lees maar hoe Frans Venter dit doen in sy jongste roman ‚Geknelde Land'. Ons huiwer dus nie om die gebruik van *fokus* as ww. aan te beveel nie.

3. Eng. *tidal drainage, tidal volume:* Die Komitee het lankal *gety-dreinering* en *getylug* hiervoor voorgestel. Nou is onder ons aandag gebring dat *wissellug* reeds in verskeie kringe in gebruik is, en ons stel dus *wissellug* vir Eng. *tidal volume* as alternatief voor. Maar *gety-dreinering* word behou vir Eng. *tidal drainage*.

4. Eng. *sedate:* Afr. *kalmeer* gee nog moeilikheid i.v.m. die *graad* waarin pasiënte gekalmeer moet word, bv. Eng. *sedate sufficiently*. Die Komitee stel voor dat

hiervoor gebruik gemaak word van bywoorde soos *voldoende, diep* e.d.m.
5. Eng. *lassitude:* Afr. *tamheid.*
6. Eng. *malaise:* Afr. behou hierdie Franse woord net so: *malaise* (spreek uit malēz).
7. Eng. *fatigue:* Afr. *moegheid* en/of *vermoeienis,* waarby dan bv. *muscle fatigue:* Afr. *spiervermoeienis.*
8. Eng. *exhaustion:* Afr. *uitgeputheid.*
9. Eng. *listlessness:* Afr. *lusteloosheid.*
10. Eng.*lethargy:* Afr. *lomerigheid* en/of *slaperigheid.*
11. Eng.*apathy:* Afr. *apatié* (met die aksent op die *ié*).
12. Eng. *euphoria:* Afr. *euforie.*

DIABETES MELLITUS IN THE URBANIZED JOHANNESBURG AFRICAN

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Diabetes mellitus is a common and important clinical problem in the urbanized Johannesburg African. A survey of Africans aged 30 and over attending the Baragwanath Hospital Casualty Department showed an incidence of diabetes of about 1%.[1] Yet, apart from Campbell's studies[2,3] on the Zulu diabetic, a search of the literature yielded little of note on diabetes in the South African Bantu. We have therefore undertaken an analysis of the records of 250 new cases of diabetes mellitus in African subjects admitted to Baragwanath Hospital during the period 1951 - 1957. These cases were selected at random and constituted at least 75% of all the new African diabetics admitted during this period.

ANALYSIS OF PERSONAL HISTORY

Age at Onset and Sex

There were 154 females and 96 males, a female predominance of 1·6:1. The distribution according to sex and age at the onset of the disease is shown in Fig. 1.

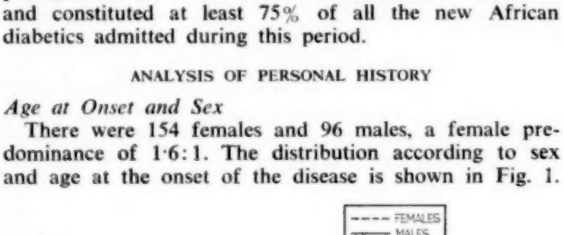

Fig. 1. Age and sex distribution of 250 African diabetics.

The majority of the patients (75·6%) were more than 40 years old when they first became ill, the peak incidence for both sexes being in the sixth decade. There was no case below the age of 12 years.

Among the patients under the age of 40 in whom constitution was specified, obese patients equalled in number those of thin or average build, while over this age obese patients predominated in a ratio of 2·7:1. Patients under the age of 40 had a higher incidence of ketosis (38%) than those over 40 (19%). The ratio of females to males was approximately the same in the age groups above and below 40.

Constitution

Constitution was specified in 184 patients (121 females and 63 males), 124 (67%) being described as obese and 60 (33%) as either thin or of 'normal', 'average' or 'medium' build. About three-quarters of the females and one-half of the males were obese.

These findings, based as they are on only a part of the total series and on the arbitrary assessment of constitution by a large number of different observers, must obviously be treated with reserve. However, an investigation of the constitution of 107 African diabetics currently attending the diabetic clinic at Baragwanath Hospital yielded essentially similar results (unpublished observations).

Of the obese patients 80% were over the age of 40, the ratio of females to males was 2·8 : 1, and the incidence of ketosis was 13%. The non-obese patients were more or less equally distributed between the different age groups, the ratio of females to males was 1 : 1, and the incidence of ketosis was 32%.

Occupation

The occupations have been divided into 2 broad categories: skilled (teachers, nurses, ministers of religion, business men, clerks, and artisans) and unskilled (manual labourers). The occupation was specified in 243 cases. Of these 14·8% were skilled workers. This is more than twice the number, 6·8%, found in a control group of admissions (100 males and 150 females). Of the male diabetics 24·7% were employed in skilled occupations as compared with 11% of the male controls. The corresponding figures for females were 8·7% and 4%.

Tribal Distribution

Sothos and Zulus, in about equal numbers, accounted for 60% of the cases. The remainder were distributed among 14 other tribes, mainly Xhosa, Tswana, Swazi, and Ndebele. A control series of 250 non-diabetic admissions to Baragwanath Hospital showed a similar tribal distribution.

Parity

In 59 postmenopausal diabetics parity was specified. These patients had a total of 334 children, an average parity of 5·66. A control group of 100 postmenopausal females drawn from a house-to-house survey in 2 Johannesburg African townships had an average parity of 5·65.[4] It is possible, however, that the parity for the diabetic women is an underestimate since in some cases stillbirths and deceased children might have been omitted from the case records.

Duration of Diabetes before Diagnosis

This was determined from the duration of the classical symptoms of diabetes (defined below). Information on only 131 cases (64 males and 67 females) was available. The

reason for this was that more than two-thirds of the total number of cases in the series presented with some complication of diabetes and specific enquiry into the duration of illness was frequently confined to the complication as opposed to the classical diabetic symptoms.

Two-thirds of the 64 males were admitted within 2 months of the onset of symptoms, whereas about one-quarter of the 67 females presented within this time. Only 11% of the males gave a history lasting longer than 1 year as opposed to 33% of the females.

MODE OF PRESENTATION

The different modes of presentation are summarized in Table I.

TABLE I. MODE OF PRESENTATION OF 250 AFRICAN DIABETICS

Presentation	*Number presenting*	*% of patients*
Classical symptoms only	54	21·6
Diabetic complications	181	72·4
Eye complications	61	24·4
Ketosis	53	21·2
Infection	40	16·0
Cardiovascular complications	9	3·6
Neurological complications	6	2·4
Miscellaneous complications	12	4·8
Incidental	22	8·8
	257*	102·8

* The excess over 250 is accounted for by 7 patients who presented with 2 complications each.

It will be noted that about 70% of the total number of cases in the series presented with one or more of the recognized complications of diabetes.

Classical Symptoms

By classical symptoms is meant polydipsia, polyuria, polyphagia, weight loss, and weakness. Only patients who volunteered one or more of these as the main complaint and who were free of any of the complications of diabetes are considered to have presented with classical symptoms. There were 54 (21·6%) such patients, 25 males and 29 females. In a further 106 patients classical symptoms were elicited. Thirteen patients (9 females and 4 males) presented with pruritus. In 7 of the females the pruritus was vulval.

Ketosis

In this analysis ketosis designates a state characterized by marked ketonuria, acidotic breathing and severe dehydration, with in most cases some degree of impaired consciousness.

Fifty-three patients (21·2%) presented with ketosis. A further 6 patients did not have ketosis on their first admission to hospital but presented with this complication on a subsequent admission. Of these 59 ketotic patients there were 27 males and 32 females. Thus 28% of the males in the series developed ketosis as compared with 20·7% of the females.

The patients in the ketotic group were younger than the patients in the series as a whole. Of the ketotic patients 40% (23 cases) were under 40 years, 44% (26 cases) were between 40 and 60 years and 16% (10 cases) were over the age of 60. In the whole series the corresponding figures were 25%, 50% and 25%.

Of the 35 ketotic patients in whom constitution was recorded, 19 (54%) were described as thin or of average build, and 16 (46%) as obese. Of the 184 cases with specified constitution drawn from the series as whole, 33% were thin or average, and 67% were obese.

Infection, mainly pulmonary, renal and cutaneous, was an additional complication in 18 (30%) of the ketotic patients. In 4 of these the patients presented with symptoms referable to both the ketosis and the infection but in the remainder the infection was discovered on examination or at necropsy.

Of the 53 patients who presented with ketosis on their first admission to hospital, 24 died. All but 1 of these died during this admission, the remaining patient dying after repeated admissions with ketosis. Of the 6 patients who presented with ketosis on a subsequent admission, 1 died.

Of the 25 ketotic patients who died (Table II, cases 1 - 25), 20 were aged 40 or more. This represents 55% of the ketotic patients in this age-group. By comparison, the mortality rate for ketotic patients under the age of 40 was 22%.

It is clear that ketosis is a common and serious complication in the African diabetic. There appear to be 2 main reasons for this high mortality. Firstly, there is the late presentation. Of the 25 ketotic patients who died, 7 were in coma on admission to hospital, 8 were in semi-coma, 6 were stuporose or mentally confused and only 4 were conscious and rational. In several instances the disturbance of consciousness had commenced 12, 24 or even 36 hours before admission. The reasons for this are mainly socio-economic and with improved living standards, educational and medical services for the African, such late presentations should become less frequent.

Secondly, in 14 of the 25 cases the ketosis was associated with some other complication which might have contributed to death. In 11 cases the additional complication was infective and in 2 it was thrombo-embolic, while the remaining case had cirrhosis of the liver with bleeding from ruptured oesophageal varices. (This latter case is described in detail under 'Deaths'). Some of these complications were probably consequences of the ketotic state, such as bronchopneumonia developing in the comatose patient, or venous thrombosis occurring with severe dehydration and hypotension; in other cases the additional complication may have served to precipitate or aggravate the ketosis.

Of the 36 ketotic patients who were discharged, about two-thirds required readmission, the main reasons being further episodes of ketosis, hypoglycaemic attacks and restabilization.

In 33 patients the insulin dosage on discharge was known. Twelve (9 males) needed more than 75 units of insulin daily, 10 (5 males) between 50 and 75 units and 11 (3 males) less than 50 units. Among the 8 women who needed less than 50 units a day there were 2 who required less than 25 units and 1 who was controlled on diet alone. It would appear that the stabilization dose of insulin is greater for the male ketotic than for the female.

Infection

Forty patients (16%) presented with symptoms referable to infection. Fifteen had pyogenic infections of the skin, and 14 pulmonary infections (3 of which were tuberculous), while the remainder manifested a variety of infections. A further 33 patients, although not presenting as such, were found to have an infection on examination. The majority of these (19 cases) had pyuria of varying degree. Of the remainder, 6 had pulmonary infections of which 4 were tuberculous, and 7 had pyogenic or fungal skin infections.

Eye Complications

Sixty-one (24·4%) patients presented with disturbances of vision. Of these, 49 had cataracts, 1 glaucoma and 1 optic atrophy. In the remainder no obvious cause could be discovered for the visual disturbance which was presumably due to metabolic changes in the lens. In a further 10 patients who presented with cerebrovascular accidents, infections or ketosis, cataracts were found on examination. Finally, there were 3 patients in whom cataracts were not observed on the first admission but were discovered on a subsequent admission.

Of these 62 cases with cataracts, 30 were over the age of 60, 28 were between 40 and 60, and 4 were under 40, the youngest being a female aged 17.

Cardiovascular Complications

Eight patients presented with cerebrovascular accidents due to haemorrhage or thrombosis and 1 with peripheral vascular disease and gangrene. On examination, 6 patients were found to have diabetic retinopathy, including 1 with massive albuminuria and hypertension. In no case was coronary artery disease detected.

In 74 patients (52 females and 22 males) the diastolic blood pressure was greater than 90 mm.Hg. In 44 patients the diastolic pressure was between 90 and 110, in 22 between 110 and 130 and in 8 between 130 and 150 mm.Hg. The majority of the hypertensive diabetics were over the age of 40 years.

Neurological Complications

Three patients presented with peripheral neuritis, 2 with myelopathy and 1 with impotence. Seven further patients were found on examination to have peripheral neuritis.

Miscellaneous

Twelve patients presented with a variety of other possible diabetic complications: stillbirths or dystocia due to large infants (6 cases), menstrual disturbance (4 cases), cholelithiasis (1 case) and necrobiosis lipoidica diabeticorum (1 case).

Incidental

In 22 cases (8·8%) the diabetes was discovered on routine examination of the urine, the patients presenting with symptoms totally unrelated to diabetes mellitus or its complications.

THERAPY ON DISCHARGE

The therapy on discharge was known in 225 patients. Diet alone controlled 14%, while 13% required between 5 and 25 units of insulin daily, 38% between 25 and 50 units, 19% between 50 and 75 units and 16% more than 75 units. Of the 32 patients controlled on diet alone, 27 were females. On the other hand, of the 35 patients requiring more than 75 units of insulin daily, 22 were males.

READMISSIONS

In the period under review 58 cases (23·2%) required readmission on 108 occasions. The reasons for readmission were hypoglycaemia due to insulin overdosage (32), restabilization (29), ketosis (16), infections (17), cataract extraction (6), injection abscesses (2), complications of pregnancy (3), and miscellaneous (3). One of the hypoglycaemic episodes resulted in permanent brain damage with dementia.

DEATHS

Thirty-four diabetic cases (23 females and 11 males) died at Baragwanath Hospital in the period reviewed. It is probable that there were further deaths at home and at other hospitals. The causes of death are summarized in Table II.

The predominant importance of ketosis is clear, being

TABLE II. CAUSE OF DEATH IN 34 AFRICAN DIABETICS

No.	Age	Sex	Constitution	Cause of death: Clinical assessment	Cause of death: Autopsy findings
1	30	M	Medium	Ketosis	Pulmonary infarction and femoral vein thrombosis
2	30	F	Obese	Ketosis	—
3	38	F	Thin	Ketosis	Bronchopneumonia
4	38	F	—	Ketosis	—
5	39	F	Obese	Ketosis	Acute bronchitis and cystitis
6	40	M	Thin	Ketosis	Nil contributory
7	40	M	—	Ketosis	—
8	42	F	Obese	Ketosis. Cirrhosis of the liver and ruptured oesophageal varices	Septal cirrhosis, varices, and marked siderosis of the liver, pancreas, spleen, heart, and stomach
9	45	F	Thin	Ketosis	Acute dilatation of the stomach
10	47	F	Obese	Ketosis	Bronchopneumonia and subacute pyelonephritis
11	48	F	Obese	Ketosis. Fibroids	Fibroids with bilateral hydronephrosis and cystitis
12	50	M	Obese	Recovery from ketosis followed by 3rd nerve palsy and contralateral hemiplegia	Mucormycosis with thrombosis of cavernous sinus and adjacent carotid artery
13	51	M	Medium	Ketosis	Nil contributory
14	52	M	Medium	Ketosis	Acute suppurative pyelonephritis
15	57	F	Obese	Ketosis. Pneumonia	—
16	57	F	Obese	Ketosis	—
17	60	M	—	Ketosis. Pulmonary embolism	—
18	62	F	Medium	Ketosis. Septic gangrene of right foot	Gangrene of right foot and calf
19	64	M	Obese	Ketosis. Anuria	—
20	67	M	Thin	Ketosis. Pneumonia	—
21	69	F	Thin	Ketosis. Gas gangrene of buttock	—
22	70	M	—	Ketosis	—
23	80	F	Thin	Ketosis	Bronchopneumonia and cystitis
24	82	F	Medium	Ketosis	—
25	50	F	—	Ketosis	Nil contributory
26	30	F	Obese	Large abscess of back complicated by ketosis	—
27	60	F	—	Stage IV carcinoma of the cervix complicated by ketosis	—
28	55	F	Obese	Gangrene of the leg; midthigh amputation with postoperative hypoglycaemia	—
29	55	F	Thin	Hypoglycaemia. Nodular hepatomegaly	—
30	66	M	Thin	? Hypoglycaemia. 'Haemochromatosis' with liver failure	Septal cirrhosis and marked siderosis of liver, pancreas, spleen, heart, adrenal, thyroid, and stomach
31	42	F	Thin	Bilateral cavitating pulmonary tuberculosis	
32	65	F	Obese	Cerebral haemorrhage	—
33	57	F	Thin	Cause of death obscure. Peripheral neuritis clinically	Generalized atherosclerosis, but no evidence of organ infarction
34	50	F	Thin	Cause of death obscure. Admitted for stabilization	—

associated with 27 of the 34 deaths. In 25 cases it occurred as the presenting feature while in the remaining 2 it complicated the course of some other presenting condition — a large abscess on the back and a stage IV carcinoma of the cervix.

Of the 7 non-ketotic deaths, 2 were due to hypoglycaemia, 1 was possibly due to hypoglycaemia in a patient with liver failure (*vide infra*), 1 was due to pulmonary tuberculosis, and 1 to cerebral haemorrhage, while in the remaining 2 the cause of death was obscure. Both hypoglycaemic deaths occurred in hospitalized patients receiving insulin. The first occurred in the postoperative period following a midthigh amputation for gangrene of the leg. In the second case hypoglycaemia developed in a patient, with an enlarged nodular liver, whose insulin requirements varied markedly.

Two cases which came to necropsy are of particular interest. The first (Table II, case 8) was admitted in semi-coma, no history being available. Examination revealed an obese, dehydrated, pale and jaundiced female with acidotic breathing. The liver was enlarged 5 fingerbreadths below the costal margin and the lower limbs were moderately oedematous. On rectal examination a melaena stool was observed. Shortly after admission the patient had 3 episodes of Jacksonian epilepsy involving the right upper limb. The blood sugar was 600 mg. per 100 ml. and the urine contained glucose ++++, and acetone ++++. A diagnosis of diabetic coma, cirrhosis of the liver, and ruptured oesophageal varices was made. On therapy the ketosis, dehydration and anaemia were largely corrected but the patient remained semi-comatose. She continued in this state for 3 days and then died after a massive haematemesis. Necropsy showed a septal cirrhosis and heavy deposits of iron in the liver, pancreas, heart, gastric mucosa, and spleen.

The second case (Table II, case 30), was admitted with backache following the lifting of a heavy weight and the diabetes was discovered on routine urine testing. In addition the patient was found to have a 4-finger enlargement of the liver, ascites, oedema of the lower limbs, and slight jaundice. A diagnosis of haemochromatosis was made. The patient was stabilized with insulin and discharged about 4 weeks after admission. Over the next 4 months he was readmitted on several occasions, 3 times for the relief of ascites and twice for hypoglycaemia. On the final admission the patient was extremely emaciated, but only moderate ascites was present. The blood sugar was 300 mg. per 100 ml. Eighteen hours after admission the patient died suddenly. The reason for this was not apparent but it may have been due to hypoglycaemia since he was given 20 units of soluble insulin 12 and 6 hours before death. Necropsy revealed a septal cirrhosis of the liver, heavy deposits of iron in the liver, pancreas, spleen, heart, adrenal, thyroid, and gastric mucosa, and slight deposits in the testes and the parotid glands.

These 2 cases thus resemble the classical picture of idiopathic haemochromatosis as described by Sheldon,[5] and are discussed later.

Apart from these 2 cases the pancreas was examined histologically in 7 cases. In 6 it was described as normal and in 1 slight atrophy of the acini was noted.

DISCUSSION

The principal characteristics of diabetes mellitus in the urbanized Johannesburg African, as shown by this study, may be summarized as follows:

The disease was commoner in females but appeared to be more severe in males as judged by their more acute onset, their higher insulin requirements on discharge from hospital and, possibly, their greater tendency to develop ketosis. The onset of the majority of cases occurred over the age of 40. No case was found below the age of 12 years. When compared with a control population a significantly greater number of African diabetics was employed in skilled occupations.

Of the cases over 40 the majority were obese and the incidence of ketosis was 19%. Those under 40 were thin, fat, or of average build and the incidence of ketosis was 38%.

About 70% of the patients presented with one of the recognized complications of diabetes — ketosis, infections, and cataracts accounting for the majority of the presentations. Ketosis was the major cause of death. By contrast myocardial infarction was absent and diabetic retinopathy, nephropathy and peripheral vascular disease were uncommon. Hypertension was frequently found but it has still to be established whether the incidence is greater than that in non-diabetic Africans.

The rate of readmission was high, the principal reasons for which were hypoglycaemic attacks, restabilization, ketosis and infection.

Other Series

The only other large series of Bantu diabetics investigated in South Africa is that of Campbell,[2,3,6] who studied Zulu diabetics in Durban, Natal. These diabetics are in the main similar to our own. About two-thirds of Campbell's cases were middle-aged, mostly overweight and insulin resistant; one-quarter were under 40, usually thin and liable to ketosis on withdrawal of insulin, while the remainder were classified as senile or peasant diabetics. The senile diabetics were generally over 60 years at onset and thin, and they usually presented with cataracts and lived in rural areas. By contrast, most of the young and middle-aged Zulu diabetics were town-dwellers. The pattern of complications was similar to that of our own cases. As regards therapy, Campbell found that his obese patients responded well to diet if this could be imposed, and also to oral antidiabetic drugs where dietary control was not possible.

In White subjects Lawrence[7] showed that diabetics are largely divisible into 2 types — the young, thin, insulin-sensitive diabetic who readily becomes ketotic when insulin is withdrawn, and the middle-aged, obese, insulin-resistant diabetic in whom ketosis is relatively uncommon. The urbanized African diabetic can, by-and-large, be similarly classified. He differs from the White diabetic principally in respect of the incidence of the vascular complications

of diabetes. In White subjects vascular disease constitutes the major diabetic complication. Of 656 deaths in diabetic subjects over the period 1950 - 1952 at the Joslin Clinic, USA, about three-quarters were due to vascular disease, mainly myocardial and cerebral infarction and diabetic nephropathy.[3] The reason for the low incidence of vascular disease in the African diabetic is unknown. It may be that in the African the diabetic syndrome is of relatively recent origin, its development being related, for example, to urbanization (*vide infra*) and that with time vascular disease may become increasingly manifest. The rarity of myocardial infarction may be related to the immunity which Africans in general exhibit towards this complication.

AETIOLOGY

Apart from such factors as age, sex and constitution, our analysis points to 2 factors as being aetiologically significant.

1. *Siderosis*

In 2 of the patients who died necropsy revealed a septal cirrhosis of the liver associated with heavy deposits of iron in the liver, spleen, pancreas, heart and gastric mucosa; in 1 of the cases there were heavy deposits in the thyroid and adrenal glands as well.

There are 2 possible explanations for these findings. Firstly, that the cases are examples in Africans of idiopathic haemochromatosis as classically described by Sheldon.[5] Secondly, that they represent in *extreme* degree the siderosis which is so common in South African Bantu of both sexes.[9-11] The qualification 'extreme' is necessary since in the average case of Bantu siderosis, the iron deposits are found mainly in the liver and reticulo-endothelial system; the heart, pancreas and other epithelial tissues are usually spared. It is suggested that in extreme cases the iron may be present in these tissues as well.

To decide between these 2 possibilities we may consider the data provided by an autopsy study on 20 African diabetics who died between 1954 and 1959.[12] Of these 20 cases, 4 (including the 2 described above) were found which were characterized by septal cirrhosis of the liver and heavy deposits of iron in the liver, spleen, heart, pancreas, and other epithelial tissues. Analysis of the 4 cases pointed strongly to their being extreme examples of Bantu siderosis rather than cases of idiopathic haemochromatosis. Firstly, if they were cases of idiopathic haemochromatosis, the number found in the autopsy series (20%) would suggest an incidence of idiopathic haemochromatosis far in excess of that described elsewhere. Secondly, 3 of the 4 cases were females; idiopathic haemochromatosis is a condition found predominantly in males. Thirdly, chemical analysis showed concentrations of iron in the spleen in all 4 cases which were considerably greater than those described in idiopathic haemochromatosis but which were similar to those found in Bantu siderosis.

2. *High Living Standards and Urbanization*

Compared with a control population, a significantly greater number of our diabetics were found to be employed in skilled occupations. Campbell[2,6] made a similar observation in his Zulu patients and showed that the diet consumed by his skilled diabetics was similar to that of White South Africans apart from a slightly higher carbohydrate intake.

The diabetics in our series were drawn from Johannesburg and the surrounding African townships. Similarly, Campbell's cases were mostly city-dwellers. That the process of urbanization is significantly related to the development of diabetes, is suggested by Campbell's[2] observation that in the obese, middle-aged Zulu diabetic (the commonest type of diabetic seen by him), there was a remarkably constant period of exposure to city life (about 20 years) before the disease became manifest.

There is also the evidence, based on survey studies,[1,13] that the incidence of diabetes in the urbanized Johannesburg African female is considerably greater than that of the rural Basuto female. The male incidence in the 2 populations was similar. However, the number of rural Basuto males surveyed was relatively small and it is possible that a more extensive study may show that, for males also, the urban incidence of diabetes is greater than the rural.

SUMMARY

An analysis of 250 cases of diabetes mellitus in the urbanized Johannesburg African is presented.

The disease was commoner in the female and in the majority of cases the onset occurred over the age of 40. In this age-group the majority of patients were obese and the incidence of ketosis was 19%. Patients under the age of 40 were of variable constitution and the incidence of ketosis was 38%.

About 70% of the cases presented with one of the recognized complications of diabetes — ketosis, infection, and cataracts accounting for the majority of presentations. Ketosis was the commonest cause of death. By contrast vascular disease, myocardial infarction in particular, was uncommon.

Attention is drawn to the possible aetiological significance of rising living standards, urbanization and siderosis.

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DIE BASAALMETABOLISME-SNELHEID VAN BLANKE- EN BANTOEVERPLEEGSTERS

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'n Verskeidenheid van faktore, sowel fisiologies as klinies van aard, beïnvloed die basaalmetabolisme-snelheid (B.M.S.). Twee van hierdie faktore, nl. klimaat en ras, is vir ons in Suid-Afrika van spesiale belang vanweë die moontlike praktiese implikasies. Op die oomblik is die standaarde waarvan ons gebruik maak uitgewerk en opgestel vir klimaatsomstandighede en rassegroepe wat aansienlik verskil van ons eie. Omdat daar aanduidings is dat die B.M.S. wel in verband mag staan met ras[1] en omdat ons hier in Suid-Afrika met verskillende rassegroepe te doen het, is dit van praktiese belang om hierdie probleem na te gaan.

Die gegewens wat hier beskryf word, is 'n voorlopige verslag van 'n reeks B.M.S.-bepalings wat op 'n ‚vergelykbare' groep Blankes en Bantoes uitgevoer is om vas te stel of daar wel plaaslik 'n korrelasie tussen ras en B.M.S. bestaan.

Omdat daar tot nog toe geen B.M.S.-standaarde vir die Suid-Afrikaanse bevolking opgestel is nie, sal die gemiddelde waardes vir Blankes en Bantoes vergelyk word met standaardwaardes wat onlangs vir die Britse bevolking uitgewerk is.[2]

PROEFPERSONE EN METODE

Altesaam 130 fisies gesonde Blanke- en 167 fisies gesonde Bantoeverpleegsters tussen die ouderdomme van 18 en 26 jaar is in die voorlopige studie as proefpersone gebruik.

Vir die doel van hierdie ondersoek is die volgende algemene prosedure deurgaans gevolg: 'n Ligte maaltyd met 'n lae proteïeninhoud is om 6·00 nm. deur die proefpersone genuttig, waarna hulle na bed is. Die saal waarin die proefpersone geslaap het, is by 'n temperatuur van tussen 20°C. en 25°C. gehou. Die volgende oggend is die suurstofverbruik om 7·00 vm. bepaal nog voordat die proefpersone opgestaan het.

Geen proefpersone is gedurende die menstruele periode ondersoek nie. Die polsspoed en mondtemperatuur van elke proefpersoon is ook vooraf bepaal. Persone met 'n abnormale hoë polsspoed of mondtemperatuur is nie in die eksperiment ingesluit nie.

Die suurstofverbruik is deur middel van twee ‚Sanborn metabulators' wat vooraf teenoor mekaar gekalibreer is, bepaal. Noukeurigheidshalwe is duplikaatbepalings op 'n reeks proefpersone uitgevoer met behulp van die ‚Sanborn metabulator' en die Douglassak-Haldane-apparaat-tegniek.[3]

Om die invloed van fisiologiese en psigologiese faktore in die toetsprosedure sover moontlik uit te skakel, is meerdere bepalings van die suurstofverbruik op elke persoon uitgevoer totdat die suurstofverbruik geen verdere afname getoon het nie. Soms was soveel as 5 of 6 bepalings nodig. Die laagste waarde wat op hierdie manier verkry is, is toe vir die berekening van die B.M.S. gebruik. Die kalorieverbruik is vanaf die suurstofverbruik bereken en wel deur aan te neem dat 4·825 kalorieë geproduseer is vir elke liter suurstof wat verbruik is.[4]

Vervolgens is die proefpersoon toegelaat om van die toilet gebruik te maak voordat die liggaamsgewig deur middel van 'n gekalibreerde Lindells-skaal bepaal is. Die naakte gewig is bereken deur 1½ lb. af te trek vir die slaapklere. Hierna is die lengte tot die naaste kwartduim gemeet. Die liggaamsoppervlakte in vierkante meters (m^2) is bereken deur gebruik te maak van die nomogram vir liggaamsoppervlakte bereken vanaf die naakte gewig en staanlengte volgens die formule van Du Bois.[5]

Die B.M.S. is bereken uit die aantal kalorieë wat per minuut verbruik is en die liggaamsoppervlakte. Die finale resultaat word dan aangegee as kalorieë/m^2/uur.

RESULTATE

In Tabel I word die gemiddelde waardes vir lengte, gewig, liggaamsoppervlakte en B.M.S., sowel as getal proefpersone in elke ouderdomsgroep vir die Blankes, aangegee. Die-

TABEL I. GEMIDDELDE LENGTE, GEWIG, LIGGAAMSOPPERVLAKTE EN B.M.S. EN DIE GETAL BLANKE VERPLEEGSTERS IN DIE VERSKILLENDE OUDERDOMSGROEPE*

Ouderdom (jaar)	*Getal*	*Lengte (duim)*	*Gewig (lb.)*	*Liggaams-oppervlakte (m^2)*	*B.M.S. (Kal./m^2/uur)*
18	40	65·8	140·1	1·68	34·3
		(2·7)	(27·4)	(0·2)	(2·3)
19	42	65·7	142·3	1·69	35·1
		(2·1)	(20·0)	(0·1)	(2·8)
20	26	65·6	135·5	1·68	35·6
		(2·3)	(18·1)	(0·1)	(2·2)
21	14	65·0	129·9	1·63	35·0
		(2·3)	(11·3)	(0·1)	(1·6)
22	4	66·3	132·6	1·67	35·0
		(1·7)	(15·1)	(0·1)	(0·9)
24	1	66·0	129·0	1·65	29·8
25	2	65·0	120·0	1·59	31·0
26	1	69·5	134·0	1·74	33·5

* Standaard-afwykings word waar moontlik tussen hakies aangegee.

selfde gegewens vir die Bantoes word in Tabel II aangegee.

Vir elke ouderdomsgroep is die Blankes en Bantoes statisties vergelyk ten opsigte van lengte, gewig, liggaamsoppervlakte en B.M.S., en wel deur gebruik te maak van die Mann-Whitney-Wilcoxon U-toets.[6] As gevolg van die onderlinge afhanklikheid van hierdie kwantiteite is 'n 2% betekenisvolle vlak aangeneem. Dus dui p-waardes wat gelyk is aan of kleiner as 0·02, statisties betekenisvolle verskille aan. Die p-waardes wat so bereken is, word in Tabel III aangegee.

Alhoewel statisties betekenisvolle verskille in lengte, gewig, en liggaamsoppervlakte in die 19-jarige groep; lengte, en liggaamsoppervlakte in die 20-jarige groep; en B.M.S. in die 21-jarige groep gevind is, kon geen deurlopende statistiese verskil vir al die kwantiteite aangedui word nie. Die gemiddelde lengte, gewig, en liggaamsoppervlakte van die Blankes was, met 'n enkele uitsondering egter, deurgaans hoër as die ooreenstemmende

TABEL II. GEMIDDELDE LENGTE, GEWIG, LIGGAAMSOPPERVLAKTE EN B.M.S. EN DIE GETAL BANTOEVERPLEEGSTERS IN ELKE OUDERDOMSGROEP*

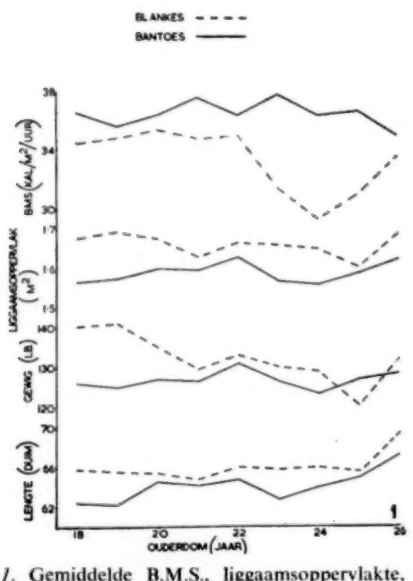
Ouderdom (jaar)	*Getal*	*Lengte (duim)*	*Gewig (lb.)*	*Liggaams-oppervlakte (m^2)*	*B.M.S. (Kal./m^2/uur)*
18	3	62·3	126·7	1·57	36·8
		(0·7)	(15·3)	(0·1)	(2·3)
19	13	62·3	124·5	1·57	35·5
		(3·5)	(16·8)	(0·1)	(2·8)
20	35	63·3	127·3	1·59	36·5
		(2·2)	(17·5)	(0·1)	(2·8)
21	39	63·4	127·0	1·59	36·8
		(1·8)	(14·8)	(0·1)	(2·6)
22	32	63·6	132·1	1·63	36·4
		(2·4)	(19·3)	(0·1)	(2·3)
23	15	62·2	126·9	1·57	37·9
		(2·2)	(13·7)	(0·1)	(3·4)
24	14	63·1	123·6	1·57	36·4
		(1·7)	(16·8)	(0·1)	(2·5)
25	10	63·8	128·0	1·60	36·5
		(3·0)	(13·5)	(0·1)	(2·7)
26	6	64·7	129·2	1·63	35·0
		(2·6)	(10·2)	(0·1)	(2·3)

* Standaard-afwykings word tussen hakies aangedui.

TABEL III. P-WAARDES BEREKEN IN DIE STATISTIESE VERGELYKING VAN BLANKE- EN BANTOEVERPLEEGSTERS IN DIESELFDE OUDERDOMSGROEPE TEN OPSIGTE VAN LENGTE, GEWIG, LIGAAMSOPPERVLAKTE EN B.M.S.*

Ouderdom	*Lengte (duim)*	*Gewig (lb.)*	*Liggaams-oppervlakte (m^2)*	*(Kal./m^2/uur)*
18	0·038	0·540	0·243	0·139
19	0·0002	0·0046	0·002	0·726
20	0·0005	0·102	0·018	0·341
21	0·026	0·545	0·200	0·018
22	0·047	0·744	0·302	0·282
25	0·364	0·758	0·910	0·044

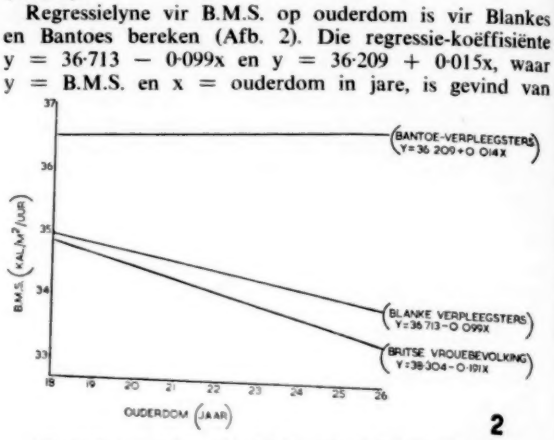
* Statisties betekenisvolle p-waardes is onderstreep.



Afb. 1. Gemiddelde B.M.S., liggaamsoppervlakte, gewig en lengte van Blanke- en Bantoeverpleegsters vir verskillende ouderdomsgroepe.

waardes vir die Bantoes, terwyl die gemiddelde B.M.S. van die Blankes deurgaans laer was as dié van die Bantoes (Afb. 1).

Regressielyne vir B.M.S. op ouderdom is vir Blankes en Bantoes bereken (Afb. 2). Die regressie-koëffisiënte $y = 36{\cdot}713 - 0{\cdot}099x$ en $y = 36{\cdot}209 + 0{\cdot}015x$, waar y = B.M.S. en x = ouderdom in jare, is gevind van



Afb. 2. Regressielyne vir B.M.S. of ouderdom vir Blanke- en Bantoeverpleegsters en vir vroue in die Britse bevolking.

toepassing te wees op Blankes en Bantoes respektiewelik. Die variasie vir die bepaling het 6·024 vir Blankes en 7·535 vir Bantoes beloop.

Die t-waarde is bereken vir die verskil tussen die regressie-koëffisiënte en beloop < 1 in absolute waarde. Die regressie-koëffisiënte vir Blankes en Bantoes verskil dus nie betekenisvol nie. Voorts is gevind dat die helling van die regressie-koëffissiënt vir Blankes en Bantoes nie betekenisvol is nie.

Die regressielyn vir Britse vroue tussen die ouderdomme 18 en 26 jaar is bereken uit die resultate van Robertson en Reid[2] en word in Afb. 2 aangetoon. Die regressie-koëffisiënt $y = 38{\cdot}304 - 0{\cdot}191x$ is van toepassing gevind. Die variasie vir die berekening het op 5·677 te staan gekom en die helling van die regressie-koëffisiënt in hierdie geval is hoogs betekenisvol ($p = {\cdot}0001$).

Die regressie-koëffisiënt vir Blanke- en Bantoeverpleegsters is voorts statisties met dié vir Britse vroue vergelyk. Geen betekenisvolle verskil tussen hierdie regressie-koëffisiënte is gevind nie.

BESPREKING

Die relatiewe klein aantal proefpersone in hierdie proefreeks, nl. 130 Blankes en 167 Bantoes, in vergelyking met meer as 1,300 Blanke vroue in die studie van Robertson en Reid,[2] regverdig geen besliste gevolgtrekking nie. Dit is egter duidelik uit die gemiddelde waardes vir lengte, gewig en liggaamsoppervlakte (Tabelle I en II en Afb. 1) dat die Bantoeverpleegsters in die huidige ondersoek 'n kleiner liggaamsbou as die Blanke verpleegsters gehad het. Voorts was die gemiddelde B.M.S.-waarde van Blanke verpleegsters vir elke ouderdomsgroep laer as dié van die Bantoeverpleegsters. Hierdie verskille was egter nie statisties betekenisvol nie. Deur 'n vergelykende studie van die antropometrie en B.M.S. van 'n groter groep Blankes en Bantoes van beide geslagte, en met 'n groter ouderdomsverspreiding, te maak, behoort die rasseverskille wat deur die huidige proefreeks aangetoon word, statisties duideliker te wees.

Uit 'n statistiese vergelyking van die B.M.S.-waardes vir plaaslike Blanke- en Bantoeverpleegsters met dié vir Britse vroue, is dit verder duidelik dat die Britse B.M.S.-standaarde vir die betrokke ouderdomsgroepe en geslag plaaslik gebruik kan word. As egter in aanmerking geneem word dat die gemiddelde B.M.S.-waarde van Bantoeverpleegsters feitlik deurgaans 2 kal./m^2/uur hoër was as die ooreenstemmende Britse waarde (Afb. 2) is dit duidelik dat 'n hoë normale B.M.S. vir 'n Bantoevrou (+ 10) moontlik abnormaal (+ 15) mag wees wanneer van die Britse standaarde gebruik gemaak word.

Dit wil dus voorkom asof die opstel van B.M.S-.standaarde vir beide Blankes en Bantoes in Suid-Afrika geregverdig is.

OPSOMMING

In 'n voorlopige studie is die B.M.S. van 130 Blanke- en 167 Bantoeverpleegsters tussen die ouderdomme 18 en 26 bepaal. Dit is gevind dat:

1. Alhoewel die gemiddelde lengte, gewig, en liggaamsoppervlakte van Blanke verpleegsters in elke ouderdomsgroep hoër was as dié van die Bantoeverpleegsters, en die gemiddelde B.M.S.-waarde van die Blanke verpleegsters vir elke ouderdomsgroep laer was as dié van die Bantoeverpleegsters, hierdie verskille nie statisties betekenisvol is nie.

2. Die B.M.S.-waardes van Blanke- en Bantoeverpleegsters statisties nie betekenisvol verskil van die standaard B.M.S.-waardes vir Britse vroue in dieselfde ouderdomsgroepe nie.

3. Die gevolgtrekking word gemaak dat die opstel van B.M.S.-standaarde vir die bevolking van Suid-Afrika geregverdig is.

SUMMARY

In a preliminary study the basal metabolic rates of 130 European nurses and 167 Bantu nurses between 18 and 26 years of age, were determined. It was found that:

1. Although the average height, weight and body-surface area of the European nurses were greater than those of the Bantu, the average BMR in Europeans was lower than that of the Bantu subjects. The differences were not statistically significant.

2. The BMR values of European and Bantu nurses did not differ significantly from the standard BMR values for British women in corresponding age groups.

3. It is concluded that the determination of BMR standards for the South African population is justified.

Die apparaat wat gedurende hierdie studie gebruik is, is deur die Nasionale Voedingnavorsingsinstituut tot ons beskikking gestel. Ons wens hiermee matrones M. White en C. Lochner van die Volkshospitaal, Pretoria, en mej. L. Lourens, Prinsipale van die Opleidingskollege vir Verpleegsters, vir hulle hulp i.v.m. die verkryging van proefpersone en akkommodasie, dr. D. J. Stoker, vir die statistiese verwerking van die resultate, en al die verpleegsters wat as proefpersone opgetree het, te bedank.

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A CASE OF TOXICITY TO EXCESS 'CARBOCAINE' WITH PROBABLE REACTIVITY OF RHEUMATIC DISEASE

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There can be no doubt that the use of local anaesthesia, where possible, is safer for the patient than the use of general anaesthesia. This is especially so in the practice of midwifery, where, as is well known, the use of general anaesthesia is attended by greater dangers and complications than in other conditions not associated with pregnancy or labour.[9,14,6] Pregnant women, particularly when in labour, are much more prone to vomit under general anaesthesia than are other patients. Inhalation of vomitus, especially the acid juices of the stomach which produce Mendelson's syndrome, is a very real and grave complication.[5] According to Eastman[4] 10% of all maternal deaths in the USA are attributable to anaesthesia, the greatest single cause of anaesthetic death being inhalation of vomitus.[13] In addition, general anaesthesia may have a depressant effect on the respiratory centre of the foetus, and further, it has an adverse effect on uterine action with an increase in the incidence of postpartum haemorrhage and manual removal of the placenta.

Hence it is not surprising that in the field of operative obstetrics, local anaesthesia has ousted general anaesthesia to a great extent. The repair of perineal tears and episiotomies, breech deliveries, low-forceps deliveries, mid-forceps deliveries (with or without manipulation of the foetal head) and even, in certain circumstances, Caesarean sections are, today, being performed with greater frequency under local anaesthesia.

Nevertheless, the use of local anaesthesia is not entirely without danger to the patient. Very occasionally one sees a severe generalized reaction to the local anaesthetic agent being used, and such a reaction can be fatal if proper and adequate treatment is not instituted immediately.

This article reports a case of severe reaction to the local anaesthetic agent used (in this instance carbocaine) and describes the interesting sequela of probable reactivation of previously quiescent rheumatic disease.

CASE REPORT

The patient, Mrs. V.D.H., was a primigravida aged 20 years. She had suffered from acute rheumatic fever at the age of

10 and again at the age of 16 years. This had left her with a mitral stenosis and aortic incompetence of mild degree.

Antenatal History

She attended the antenatal clinic at the Queen Victoria Hospital, Johannesburg, from 22 September 1959, when she was 11 weeks' pregnant. Her menstrual cycles had always been regular (C.13 3-4/21-22). Her last menstrual period was on 8 July 1959 and the expected date of delivery was 14 April 1960. She was referred to the Cardiac Clinic for assessment and the diagnosis of mitral stenosis and mild aortic incompetence was confirmed. She was regarded as a cardiac Type 1 case and was given a good prognosis for this pregnancy.

At 12 weeks' gestation she was admitted with mild hyperemesis gravidarum. This was soon controlled and she was discharged after a few days' treatment. Thereafter she attended the antenatal clinic regularly, paying 16 visits throughout her pregnancy. Her blood pressure varied between 110 and 130 mm.Hg systolic and 40 and 60 mm.Hg diastolic. At no stage was albuminuria present. Slight oedema was found on 2 occasions and her weight had increased from 124 pounds at 11 weeks to 148 pounds at 37 weeks. Her cardiac status remained unimpaired.

Labour

She was admitted to hospital at 37 weeks' gestation for reassessment of the cardiac state and for rest before labour. She commenced labour spontaneously at 6.45 a.m. on 11 April 1960, when she was pregnant for 39 weeks and 4 days according to her dates. The first stage of labour lasted 8 hours 25 minutes and progressed normally. At the beginning of the second stage the vertex was found to be 1 inch below the ischial spines with the sagittal suture in the direct anteroposterior diameter. After 10 minutes in the second stage, with good contractions and bearing-down efforts, there was no appreciable advance of the head. Since the patient had a cardiac lesion and since some slight meconium-staining of the liquor was noted, it was decided to apply low forceps under a pudendal block. Forty ml. of 2% carbocaine (without adrenaline) were used in performing the pudendal block. Anderson's forceps were applied, an episiotomy was done and an easy low-forceps extraction was performed. The infant was a male, weighing 8 lb. 9 oz., and was alive and well. The second stage had lasted 25 minutes and the third stage lasted 5 minutes, the placenta delivering spontaneously with a total blood loss of 250 ml.

Severe Reaction and Treatment

Approximately 15 minutes after the pudendal block was given and just after the third stage had been completed, the patient was seen to become very excitable, restless, emotional and almost maniacal. This excitable phase lasted about 30 seconds. She then commenced to twitch, developing typical Jacksonian movements in her left arm. These progressed to generalized epileptiform convulsions. Morphine, gr. ¼, was given intravenously soon after the fit had ended and paraldehyde, 8 ml., was injected intramuscularly. The convulsions recommenced almost immediately after these injections and in a relatively short time the patient had about 10 convulsive seizures in rapid succession. The outstanding feature was the high level of excitability, in that the slightest stimulus precipitated another fit. A general anaesthetic was administered and the fits were controlled.

The patient was found to be cold and clammy and quite pulseless, manifesting the signs of severe shock. One ampoule of 'levophed special' was given intravenously and an intravenous drip containing 100 mg. of 'solucortef' was begun. On this regime her general condition improved, though the blood pressure fluctuated between 90/50 and 120/60 mm.Hg for the next 1½ hours, after which it remained at about 120/60 mm.Hg. The heart rate when the anaesthetic was begun was 140 per minute, but this gradually dropped to about 116 per minute and remained at this rate for the next 4 hours. For a short time, while she was under the anaesthetic, there was evidence of mild pulmonary oedema in the form of bilateral crepitations and moist bronchiolar sounds, but these signs disappeared and did not recur. A catheter specimen of urine taken just after the start of the anaesthetic had revealed a faint trace of albumin on the cold test but no albumin on the hot test. Albuminuria, however, was definitely present in the next specimen of urine, obtained 3 hours after the fits had occurred. All specimens of urine obtained thereafter were normal, and albuminuria was not found again. This one instance of albuminuria was probably due to the series of fits that occurred.

On several occasions an attempt was made to discontinue the general anaesthetic, but, as soon as the level of anaesthesia dropped, twitching of the limbs was seen, indicating that convulsions would probably recur if the anaesthetic was stopped. There were no obvious localizing signs in the central nervous system and the ECG showed only a tachycardia and some signs of left auricular enlargement. The uterus remained well contracted. 'Somnifaine', 1 ml., was given intramuscularly about an hour after the original convulsion and this was repeated again an hour later.

Progress and Diagnosis

The anaesthetic was finally stopped 2¼ hours after it had been started. There was no twitching of muscles and the patient's condition appeared satisfactory, though she was still unconscious. She was nursed in an oxygen tent and close watch was kept on her blood pressure, pulse rate and respirations. The patient regained consciousness 3¼ hours after the anaesthetic was stopped and 5½ hours after the series of convulsions. She appeared quite normal and was orientated as to time, place and person. Her blood pressure was 120/60 mm.Hg, the lung fields were clear, her pulse rate was 108 per minute and there were no abnormal signs in the central nervous system. Her good general condition was maintained thereafter.

When the first convulsive episode occurred, the immediate diagnosis that was considered was that of postpartum eclampsia. However, on further consideration this seemed unlikely, since at no stage in pregnancy or labour had the patient shown any signs of pre-eclampsia. When the state of severe shock developed, a sensitivity reaction to a rather large dose of the local anaesthetic agent was considered and was found to be the most feasible explanation for the convulsions and the collapse. A possible amniotic embolization or, as the patient had a cardiac lesion, a cerebral embolism, or even a cerebral haemorrhage, was entertained in the differential diagnosis but, as the case developed, these appeared more and more unlikely. The diagnosis, therefore, that appeared most likely to be correct, was that of sensitivity to the local anaesthetic agent, and the subsequent clinical picture appeared to confirm this diagnosis.

Rheumatic Fever Exacerbation

On the sixth postpartum day the patient was found to have a painful and swollen right knee joint and some effusion was also found in the left knee joint. There was no throat infection but the lochia was seen to be somewhat offensive. The swollen joints were thought to be due to a possible exacerbation of rheumatic fever resulting from the violent movements of the convulsive state, and 'disprin', grs. 10 *t.d.s.*, was prescribed. The following day she complained of pain in both ankle joints and these were found to be swollen as well. No other joints appeared involved. A pyrexia of 101° F. and a tachycardia of 110 per minute were recorded. She was given a broad-spectrum antibiotic and the disprin was continued. The pyrexia and tachycardia persisted for 3 days and then returned to normal levels.

The question whether the pyrexia and tachycardia were due to a rheumatic fever exacerbation or to a mild uterine sepsis was discussed, but in view of the swollen and painful knee and ankle joints, the senior physician considered it wiser to regard the episode as being due to a rheumatic exacerbation. Laboratory tests were of no help since the results obtained might normally have been found in the puerperium. We considered whether the swollen joints could have resulted purely from the violent movements of the convulsions *per se*, but since these joints were perfectly normal for the first 5 days of the puerperium this seemed unlikely.

The cardiac state remained unaffected. The pain and swelling

of the joints regressed gradually, the ankle joints returning to normal first. By the 22nd puerperal day there was no longer any pain in the knee joints but there was still slight excess fluid present. When this patient was seen at the postnatal clinic, 6 weeks after her delivery, she was fit and well and her knee joints had returned to normal.

DISCUSSION

The use of local anaesthesia is an eminently safe and satisfactory procedure, but in a very small proportion of cases a severe reaction to the local anaesthetic agent is seen. This possibility is well recognized and documented. Martin[12] discussed a case of reaction to procaine, while Dutton,[3] Barnes,[1] Lock and Greiss,[11] Bennett,[2] and Goldman[7] reported reactions following the injection of xylocaine ('lignocaine'). Reports on the use of carbocaine are still scanty and, to our knowledge, no account appears in the literature of a convulsive reaction occurring after the use of carbocaine.

Adverse reactions to most types of local anaesthetics appear to have a basically common cause and pattern. The causes may be divided into two groups: (*a*) toxic, and (*b*) idiosyncrasy.

Toxic Reactions to Local Anaesthetics

The toxic group comprises by far the largest number of patients who react to the local anaesthetic agent. Here the symptoms and signs are caused by too high a concentration of the local anaesthetic in the blood stream. Normally, the local anaesthetic agent is broken down in the blood stream and in the liver, and whenever the amount of local anaesthetic agent is in excess of that which the patient's body can deal with at any one time, toxic reactions will occur. The exact blood concentrations that bring about toxic reactions will vary from patient to patient, but drug manufacturers do impose a limit on the amount of local anaesthetic agent that should be used, this amount being well within the range in which the body can break down the agent sufficiently quickly to prevent reactions.

Special care must be exercised, therefore, to avoid conditions where too high a concentration of the local anaesthetic agent in the blood stream might be brought about. The first and most obvious of these is, of course, not to use more than the recommended amount of local anaesthetic. In the case of procaine and xylocaine the generally accepted maximum dose is 1 g., i.e. 50 ml. of a 2% solution, 100 ml. of a 1% solution or 200 ml. of a 0·5% solution. In a personal communication, A. B. Bofors (Sweden), the manufacturers of carbocaine, have informed us that the maximum permissible dose of carbocaine is 0·2 g. when used without a vasoconstrictor, or 0·5 g. when used with a vasoconstrictor such as adrenaline. The makers, however, point out that doses twice or even 3 times their recommended maximum dose have often been used without toxic effects. In the case reported above, 40 ml. of a 2% solution of carbocaine were used without the addition of adrenaline. Thus 0·8 g. of the local anaesthetic agent was used – which is 4 times the recommended dose without a vasoconstrictor, and this must obviously have been the cause of the toxic reaction seen.

Too high a blood concentration of the local anaesthetic might also be produced by inadvertently injecting the anaesthetic agent into a vein during infiltration of the tissues. Hence it is essential to keep the needle moving or to draw back on the plunger of the syringe if a large amount of anaesthetic agent is to be injected in one area. This is particularly important when injecting into a very vascular area, such as the perineum, during pregnancy and labour, when the blood supply is very rich. This increased vascularity in itself would cause the anaesthetic agent to be absorbed into the blood stream much faster than normal. Finally, the use of such spreading agents as 'hyalase' (hyaluronidase) should not be used, since, with the potent spreading effect of hyalase, greater and faster absorption of the local anaesthetic takes place.

Idiosyncrasy to Local Anaesthetics

Idiosyncrasy to the local anaesthetic being used is rare, and Hawksworth[8] is even doubtful whether it ever occurs. However, since there are many reports of reactions to local anaesthetics where doses well within the recommended limits have been used, such a condition of idiosyncrasy or drug sensitivity to the local anaesthetic agent must be accepted. This state is one peculiar to the individual patient, and such a patient should be warned specifically against the possible use of that agent in the future.

Signs and Symptoms of Toxicity

Toxic reactions to local anaesthesia may be commoner than is generally thought, the milder reactions often being attributed to the patient's nervousness or excessive anxiety.

The toxic effect on the central nervous system is usually twofold. There is a stimulatory effect as shown by restlessness, loquacity, psychic disturbances,[7] vomiting, twitching of muscles, tremors, and finally generalized convulsions. A depressive effect on the vasomotor and respiratory centres in the medulla is seen, as shown by a sudden and marked drop in the blood pressure and respiration. Sudden inability of the patient to speak should be regarded as a grave symptom, since it heralds unconsciousness.[10] A typical generalized anaesthetic state then ensues which may be followed by death if vigorous and adequate treatment is not given immediately.

Treatment

The aim of treatment is to control the stimulatory effects on the central nervous system and to counteract the depressive effects on the vasomotor and respiratory centres. The short-acting barbiturates are generally accepted as being specific for the control of convulsions and these are give intravenously in hypnotic doses. Sodium thiopentone would appear to be the drug of choice for this purpose.

Cardiovascular collapse should be treated by intravenous stimulants such as 'methidrine' in doses of 10 mg., by setting up an intravenous drip containing 'levophed' if necessary, or by giving 'levophed special', which can be given intravenously without dilution. The efficacy of this treatment will, of course, be judged by the blood pressure.

Depression of the respiratory centre will cause marked slowing or even cessation of respiration. It then becomes necessary to maintain respiration by artificial means. This is best achieved by passing an intra-tracheal tube if the patient is unconscious or, at least, an airway, and ensuring

an adequate oxygen supply by the use of the reservoir bag of an anaesthetic gas machine. This maintainence of respiration is possibly the most important part of the treatment.

CONCLUSIONS

The following suggestions are made with a view to reducing or avoiding toxic reactions to local anaesthetic agents:

1. The maximum dose of these drugs must be respected. The smallest amount in the lowest effective concentration should always be used.

2. An aspiration test for blood must always be made. When large amounts of local anaesthetic are being injected over a large area, the needle should be kept moving.

3. The addition of a vasoconstrictor drug, such as adrenaline 1:1,000, to give a final concentration of 1:200,000 in the local anaesthetic solution injected, is recommended. Conversely, the use of spreading agents such as hyalase, should be avoided.

SUMMARY

A case of a toxic reaction to excess carbocaine is described. The patient had had rheumatic fever and an exacerbation of this disease occurred after the severe central-nervous-system stimulation of the reaction.

The differences between toxic reactions and idiosyncrasy to local anaesthetics are discussed and treatment is described in some detail.

Suggestions for reducing or avoiding toxic reactions to local anaesthetic agents are made.

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43rd MEDICAL CONGRESS (M.A.S.A.), CAPE TOWN, 24 - 30 SEPTEMBER 1961 : 43ste MEDIESE KONGRES (M.V.S.A.), KAAPSTAD, 24 - 30 SEPTEMBER 1961

WETENSKAPLIKE UITSTALLINGS

'n Omsendbrief van die Voorsitter van die Sub-Komitee vir Wetenskaplike Uitstallings word hier weergegee ter inligting van lede van die Vereniging:

„Dit is die voorneme van die Organiserende Komitee om 'n wetenskaplike uitstalling te reël in aansluiting by die verrigtinge van die kongres. Ons voel dat dit 'n ongeëwenaarde geleentheid sal bied vir die demonstrasie van wetenskaplike navorsing en ander werk wat tans in die Unie onderneem word, nie alleen deur offisiële liggame en ondersteunde navorsingseenhede en groepe nie, maar ook deur indiwiduele dokters en wetenskaplikes.

„Die benadering van hierdie uitstalling sal so wyd as moontlik wees en sal die volgende insluit: antropologie en genetika, sielkunde en industriële toetse vir geskiktheid, openbare gesondheid en sanitasie, die mediese toepassing van atoomkrag, voeding, voedingstegnologie en kwaliteitsbeheer, terapeutiese stowwe, produksie en beheer, gesondheidsonderrig en immuniteitsmetodes, veeartsnykundige werk en die soonose, hospitaalbeplanning en die administrasie van mediese diens, die geskiedenis van medisyne en die instrumente en stamgewoontes, asook die hele gebied van die mediese navorsing. Algemene praktisyns word ingesluit.

„Een deel van die uitstalling sal 'n bioskoop wees wat 'n daaglikse program van mediese- en wetenskaplike films sal vertoon, kleurskyfies, ens. Daar word gehoop dat geslote beeldradio ingesluit sal word.

„Hierdie voorlopige omsendbrief word aan so veel organisasies en afsonderlike persone moontlik gestuur wat moontlik wil deelneem aan die uitstalling. Ten einde die beplanningskomitee te help, sal dit waardeer word indien voornemende uitstallers sal aandui — so spoedig moontlik — of hulle of hulle organisasies gewillig is om deel te neem aan die uitstalling en, indien wel, dui asseblief kortliks aan:

„1. Aard en titel van die uitstalling.

2. Of dit (*a*) staties sal wees, of (*b*) 'n werkende demonstrasie, of (*c*) film- of kleurskyfie-projeksie.

3. By benadering wat (*a*) die lengte, of (*b*) die vierkantvoetsmeting of uitstallingsruimte benodig, sal wees.

4. Indien krag, water, dreinering, of gas verlang word.

5. Indien films of skyfies vertoon sal word, die tyd wat dit ongeveer in beslag sal neem.

„Uit hierdie inligting sal voorlopige planne opgestel word en 'n opvolgingsbrief vroeg gedurende 1961 sal handel oor die besonderhede, planne, reëlings vir demonstrateurs, aflewering en oprigting, versekering, ens.

H. O. Hofmeyr
Voorsitter, Sub-Komitee vir Wetenskaplike Uitstallings

Mediese Huis
Waalstraat 35
Kaapstad

PASSING EVENTS : IN DIE VERBYGAAN

South African Paediatric Association (M.A.S.A.), Paediatric Prize Essay 1961. The subject for the 1961 Prize Essay is 'The investigation and treatment of recurrent abdominal pain in children over six years of age'. Entry is open to all 5th and 6th year medical students in South Africa. The essay must be limited to 5,000 words, may be in English or Afrikaans, and must be typed in double spacing, and submitted together with one copy. Entries should be sent to the Hon. Secretary, South African Paediatric Association, Department of Child Health, University of Cape Town, Medical School, Observatory, Cape, before 1 May 1961.

The prize is a Bronze Medal and the sum of £10 0s. 0d. for the purchase of books, instruments or subscriptions to journals.

The Paediatric Association reserves the right, if in any year there is in the opinion of the examiners no essay of sufficient merit, to make no award for that year.

* * *

Dr. H. J. H. Claassens, obstetrician and gynaecologist, of Cape Town, has changed his residential telephone number. His home number will now be 2-4640 and is not listed in the current Telephone Directory.

Dr. H. J. H. Claassens, verloskundige en ginekoloog, van Kaapstad, het sy woning se telefoonnommer verander. Sy huisnommer is nou 2-4640 en verskyn nie in die jongste Telefoongids nie.

Correction. An error occurred in the Discussion of the article 'Moonflower poisoning' which appeared in the issue of the *Journal* for 31 December 1960 (**34**, 1114). In the last paragraph of the Discussion the third, fourth, and fifth lines should read: 'It may be either *Datura cornigera* Hook or *Datura arborea* L., but if these prove to be con-specific the correct name for the complex will be the former'.

The authors were Drs. Joan Griffiths and D. P. Gordon Smith. The second author's name was given in error as 'D. J. Gordon-Smith'.

THE BENEVOLENT FUND : DIE LIEFDADIGHEIDSFONDS

The following donations during December 1960 are gratefully acknowledged:

Met dank word die volgende skenkings gedurende die maand Desember 1960 erken:

Votive Cards in Memory of: Geloftekaarte ter Nagedagtenis aan:

Dr. L. Mirvish *by* Drs. R. Schaffer and Dr. S. M. Geffen; Dr. A. Simpson-Wells *by* Dr. J. Abelsohn; Dr. H. Pearson *by* Dr. D. Ferguson and family, Dr. A. Ferguson, Mr. Claude Le Fevre, Mr. H. D. Duncan, Mr. D. R. Smith, Miss E. F. Horne, and Dr. J. O. Pearson (Alex Sharwood (Pty.) Ltd.); Dr. J. H. Sypkens *by* President and members of Northern Transvaal Branch (M.A.S.A.); Dr. H. Goldin *by* President and members of Northern Transvaal Branch (M.A.S.A.), Drs. McLachlan, Smith and Macdonald; and Dr. A. C. Watt *by* Drs. R. W. S. Cheetham and M. S. Paterson.

Total Received from Votive Cards: £21 10s. 6d.
Totaal Ontvang van Geloftekaarte:

Services Rendered to: Dienste Gelewer aan:

Dr. A. Lawson *by* Drs. Navid, McCabe and Bloch.
Mr. and Mrs. E. Connellan *by* Dr. R. Schaffer.
Duma Gcabashe *by* Dr. Harvey Illing.
Dr. F. D. Pascoe *by* Mr. H. C. Warner, Drs. R. J. Venning and R. R. McKenzie.
Mrs. G. F. Campbell *by* Prof. F. Forman and Dr. V. Brink.

Total Received from Services Rendered: £37 17s. 0d.
Totaal Ontvang van Dienste Gelewer:

Donations : Donasies:

	£	s.	d.
Dr. D. K. Bowden and Mrs. Bowden	4	4	0
Dr. D. Lipschitz	3	3	0
Drs. Jurgens, Schultz, Krynauw and van der Berg	10	10	0
Mr. L. J. G. Kruger	4	4	0
Dr. and Mrs. N. J. Terblanche	4	4	0
Dr. H. Taylor-Smith	3	3	0
Dr. D. S. Gous and C. W. Haveman	6	0	0
Drs. P. H. Marks and S. J. Lazar	6	6	0
Dr. N. Fram	4	4	0
Drs. Effren, Kuny and Sacks	6	6	0
Drs. Fram, Gould, Bostock and Silansky	5	5	0
Drs. Thomas, Bruschi and Wright	10	10	0
Drs. Harries, Kuschke and Hofmeyr	1	1	0
Dr. Lucy Davis — 'Kindness from Dr. J. Anderson'	2	2	0
Cape Western Branch (M.A.S.A.) collection box	2	13	0
Cape of Good Hope Faculty of the College of General Practitioners and Groot Drakenstein Division (M.A.S.A.)	4	10	0
Cape Western Branch (M.A.S.A.) — Balance, proceeds of Film Première	84	3	7
Dr. J. H. Botha		10	6

Total Received from Donations: £162 19s. 1d.
Totaal Ontvang van Donasies:

Grand Total: Groot Totaal: £222 6s. 7d.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

SULFAPLASTIL

Newport Trading Corporation (Pty.) Ltd., announce the introduction of Sulfaplastil suspension, an absorbent and flexible skin plastic film, manufactured by Dr. Mann Laboratories, and supply the following information:

Sulfaplastil contains dimethyl-pyrimidine-sulfanilamide and acetyl-amido-4-aminobenzylsulfonamide in a plastic vehicle of polyvinyl pyrrolidin. It is a rational topical sulphonamide treatment of all infective lacerations or wounds, cuts, and burns which, by sealing off the trauma, aims at the prevention of further spreading of the infection and assists local defence mechanisms of the leucocytic system before this becomes active.

It is recommended that 2 - 3 applications per day is sufficient.

Further information may be obtained from Newport Trading Corporation (Pty.) Ltd., P.O. Box 1871, Johannesburg.

INTRAVAL 750 MG. SUPPOSITORY

Maybaker (S.A.) (Pty.) Ltd. announce the addition of a 750 mg. suppository to their range of Intraval brand thiopentone sodium suppositories.

The complete range now consists of suppositories each containing 125 mg., 250 mg., 500 mg. or 750 mg. thiopentone sodium. The suppositories are indicated as a basal narcotic in pre-anaesthetic medication in children. The suggested method of use is to administer the suppositories rectally 15 - 30 minutes before anaesthesia in a dosage of 20 mg. per kg. bodyweight.

Further particulars may be obtained from the Medical Department, Maybaker (S.A.) (Pty.) Ltd., P.O. Box 1130, Port Elizabeth.

NEO-CHLORAL-PED

Westdene Products (Pty.) Ltd. announce that Neo-Chloral-Ped, a safe mild hypnotic for children, is now available in tablet form, and supply the following information:

Neo-Chloral-Ped is a product of South African research manufactured by Pediatric Laboratories (Pty.) Ltd., Cape Town. It contains a complex organic salt of chloral-hydrate possessing no unpleasant flavour whatsoever. It is a safe, mild hypnotic giving a natural sleep from which children can be easily awakened. There are no after-effects and painful conditions are not masked.

Neo-Chloral-Ped has been available for some time as an elixir and the increasing demand for this has encouraged the introduction of a tablet form for older children.

Further information may be obtained from the sole South African distributors, Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

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Body Fluids in Surgery. 2nd edition. By A. W. Wilkinson, Ch.M., F.R.C.S.E., F.R.C.S. Pp. xi + 276. 21s. net + 1s. 5d. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1960.

An Introduction to Congenital Heart Disease. By Leo Schamroth, M.B., B.Ch. (Rand), M.R.C.P.E., F.R.F.P.S. and Fay Segal, M.D. (Rand). Pp. ix + 116. 82 illustrations. 22s. 6d. net. Oxford: Blackwell Scientific Publications Ltd. 1960.

Rose and Carless Manual of Surgery. 19th edition. Consulting Editor: Cecil Wakeley, Bt., *K.B.E., C.B.*, LL.D., M.Ch.,

D.Sc., F.R.C.S., F.R.S.E., F.A.C.S., F.R.A.C.S. Pp. xv + 1389. Illustrated (12 colour plates). 84s. London: Baillière, Tindall and Cox Ltd. 1960.

The Principles and Practice of Chiropody. 2nd edition. By John H. Hanby, F.Ch.S. and H. E. Walker, F.Ch.S. Pp. xii + 433. 151 figures. 30s. London: Baillière, Tindall and Cox. 1960.

Clinical Tropical Diseases. 2nd edition. By A. R. D. Adams and B. G. Maegraith. Pp. x + 540. 72 illustrations. 52s. 6d. net. Oxford: Blackwell Scientific Publications Ltd. 1960.

Diagnosis in Locomotor Disorders. By Kenneth Stone, D.M. (Oxon), M.R.C.P. Pp. 224. 53 illustrations. 25s. London, New York and Toronto: Oxford University Press. 1960.

Surgery and Clincial Pathology in the Tropics. By Charles Bowesman, *O.B.E.*, B.A., M.D., F.R.C.S.E., F.A.C.S., D.T.M. & H.Ed. Pp. viii + 1068. 321 illustrations. £5 10s. 0d. plus 5s. postage abroad. Edinburgh: E. & S. Livingstone Ltd. 1960.

Peptic Ulceration — A Symposium for Surgeons. By Charles Wells and James Kyle, M.B., M.Ch., F.R.C.S., F.R.C.S.(I.). Pp. xi + 260. 44 illustrations. 42s. net. + 2s. 1d. postage abroad. Edinburgh: E. & S. Livingstone Ltd. 1960.

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Fluid Balance in Obstetrics. By Philip Rhodes, M.A., M.B., F.R.C.S. (Eng.) M.R.C.O.G. Pp. x + 169. 25s. net. London: Lloyd-Luke (Medical Books) Ltd. 1960.

Leukaemia — Research and Clinical Practice. By F. G. J. Hayhoe, M.A., M.D. (Cantab.), M.R.C.P. (Lond.). Pp. viii + 335. 12 coloured plates and 196 black-and-white illustrations. 80s. net. London: J. & A. Churchill Ltd. 1960.

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Aids to Biochemistry. 5th edition. By S. P. Datta, B.Sc., M.B., B.S. and J. H. Ottaway, B.Sc., Ph.D., A.R.I.C. Pp. 264. 29 tables, 41 illustrations. 15s. + 1s. 3d. postage. London: Baillière, Tindall and Cox. 1960.

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The Antibiotic Saga. By Henry Welch, Ph.D. and Félix Martí-Ibáñez, M.D. Pp. 150. $3·00. New York: Medical Encyclopedia, Inc. 1960.

Insecticide Resistance and Vector Control. Tenth report of the Expert Committee on Insecticides. Technical Report Series No. 191. Pp. 98. 5s. 1d. Also in French and Spanish. South African agents: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724, Pretoria. Geneva: World Health Organization. 1960.

A Synopsis of Fevers and their Treatment. 10th edition. Revised by James H. Lawson, M.D. (Glas.) D.P.H. Pp. viii + 184. 10s. 0d. London: Lloyd-Luke (Medical Books) Ltd. 1960.

General Anaesthesia. 2 volumes. Edited by Frankis T. Evans, M.B., B.S., F.F.A.R.C.S., D.A. and Cecil Gray, M.D., F.F.A.R.C.S., D.A. Vol. 1 — *Basic Principles;* Vol. 2 — *Techniques, Special Fields and Hazards*. Vol. 1 — pp. xii + 531 + 29. 121 figures. Vol. 2 — pp. xiii + 431 + 29. 78 figures. £8 1s. 9d. plus 2s. 9d. postage. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1959.

Radiology as a Diagnostic Aid in Clinical Surgery. By Howard Middlemiss, M.D., F.F.R., D.M.R.D. Pp. viii + 151. 86 illustrations. 30s. 0d. net. London: William Heinemann (Medical Books) Ltd. 1960.

Modern Trends in Accident Surgery and Medicine. Edited by Ruscoe Clarke, *M.B.E.*, M.B., F.R.C.S. (Eng.); F. G. Badger, B.Sc., F.R.C.S. (Ed.) and Simon Sevitt, M.D., M.Sc., F.R.C.P.I., D.P.H. Pp. xi + 330 + 16. 82 figures. £4 4s. 6d. plus 2s. 0d. postage. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1959.

Surgical Aspects of Medicine. Edited by H. Daintree Johnson, M.A., M.B., B.Chir., F.R.C.S. Pp. xv + 382 + 21. £3 13s. 0d. plus 1s. 9d. postage. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1959.

Year Book of Urology, 1959 - 1960. Edited by William Wallace Scott, M.D., Ph.D. Pp. 374. 79 figures. $8·00. Chicago: Year Book Publishers, Inc. 1960.

BOOK REVIEWS : BOEKBESPREKINGS

SEWARD'S BEDSIDE DIAGNOSIS

Bedside Diagnosis. 5th edition. By Charles Seward, M.D., F.R.C.P. (Edin.). Pp. xix + 479. 25s. net + 1s. 5d. postage abroad. Edinburgh: E. & S. Livingstone Ltd. 1960.

Five editions in English with one reprint, two editions in Spanish, and one in Portuguese, show something of the popularity of this useful book. In this edition several sections have been rewritten, new advances in diagnostic methods have been incorporated and a new section on 'Some uses of radioactive isotopes in diagnosis' by Dr. Mark Ridley has been added, as well as one on 'Drugs considered as causes of symptoms'.

The author acknowledges the help and advice of a number of experts in bringing his work up to date, and in his foreword Lord Cohen of Birkenhead writes, 'The student who masters the principles on which this handbook is based will have an intelligent and rewarding approach to the diagnosis of disease, and he will have laid a foundation which will remain firm whatever stress the superstructure of later knowledge may impose upon it. And even the experienced practitioner will learn much from its text'. Experience has shown that his estimate is undoubtedly correct. A.H.T.

BRUCELLA INFECTION IN MAN

Brucella Infection and Undulant Fever in Man. By Sir Weldon Dalrymple-Champneys Bt., *C.B.*, D.M., D.P.H., F.R.C.P. Pp. 196. 20 illustrations. London, New York, Toronto: Oxford University Press. 1960.

As late Deputy Chief Medical Officer, Ministry of Health, London, the author presents an informative review of the first 1,500 indigenous cases of undulant fever (excluding laboratory infections) he investigated since 1929. This forms the clinical basis of his present work, a complete and concise book on brucella infection in man.

In Great Britain, the drinking of cow's milk has accounted for the vast majority of human cases.

Brucella is rarely the sole cause of death but causes much misery and loss of work. The correct diagnosis is often missed by the doctor, and the patient is then liable to the tragedy of being labelled neurotic.

Despite modern antibiotics, the object of treatment is rather to establish or re-establish a comfortable symbiosis than to eradicate the parasite at any cost.

In this book a great deal of information on brucellosis is collected, (even transmission by blood transfusion), which makes it a valuable reference work for student, general practitioner, bacteriologist, physician, and health official. J.S.L.

YEAR BOOK OF DRUG THERAPY

The Year Book of Drug Therapy, 1959 - 1960. Edited by Harry Beckman, M.D. Including a new section on *Evaluation of the Year's New Drugs.* Pp. lxxx + 570. $8.50. Chicago: The Year Book Publishers, Inc. 1960.

The Year Book series is to be found in all medical libraries, and according to their general or particular interest special volumes are regularly ordered by many medical practitioners. The annual volume dealing with drug therapy is obviously one of great importance. The abstracts provide information on all sorts of drugs currently in the melting pot. The articles which have been reviewed have appeared in a variety of journals, few of which are seen by the ordinary reader. The literature appearing up to September 1959 has been sifted, and selected articles quite fully reproduced. Such recently introduced drugs as darenthin, ritalin, tofranil, diabinese, decadron, fungizone, and numerous others are considered here. References to older drugs are also to be found, sometimes in re-appraisal, sometimes in new therapeutic applications. There are the traditional hard-hitting editorial comments, and full author and subject index. A new feature is a section of about 80 pages printed on blue paper and with its own index, in which 334 trade preparations are briefly evaluated. This is a most useful addition to this volume, which has for years proved most valuable. N.S.

HAND INJURIES

Surgery of Repair as Applied to Hand Injuries. 2nd edition. By B. K. Rank, *C.M.G.*, M.S. (Melbourne), F.R.C.S. (England), F.R.A.C.S. and A. R. Wakefield, M.S. (Melbourne), F.R.C.S. (England), F.R.A.C.S. Pp. xiv + 284. 219 figures. 45s. net. Edinburgh and London: E. & S. Livingstone Ltd. 1960.

The surgery of the hand is at present in the forefront of surgical thought and endeavour, and rightly so. As a result of the work of Bunnell, Pulvertaft, Furlong and others, tremendous strides have been made towards ensuring an optimum return to function of the injured hand. In spite of mechanization and automation there is an increasing demand for manual dexterity. The specialized techniques and skills for reconstructing these badly maimed hands are in constant demand.

This book, within the limits of its title, provides an excellent ready answer to these demands. The new edition of this widely acclaimed work has changed but little from the previous one. The authors are obviously surgeons of wide experience, skill and knowledge, as judged by their writings. The highest tribute the reviewer can pay them, after having read this book, is to say that he would like to have their services for himself, if he suffered a serious hand injury.

Their fascinating book is strongly recommended to all surgeons who deal with hand injuries. A.S.

DIABETES

Diabetes. With a chapter on *Hypoglycemia.* By 54 authors. Edited by Robert H. Williams, M.D. Pp. xvi + 793. 192 illustrations, 23 in full colour, $20.00. New York: Paul B. Hoeber, Inc. 1960.

This is a full and nicely produced book on diabetes, written by a large number of authors who are experts in their own fields. It is a suitable companion volume to 'Joslin', being written far more from the scientific and laboratory angle than from the clinical and social.

It was very disappointing to the reviewer to find so little on the fundamental problem of latent diabetes, prediabetes, the relationship to obesity, and the features of the babies of the diabetic and of the prediabetic. The relationship of pregnancy to diabetes is very inadequately considered. The remarkable pancreas of the diabetic's baby, which may contain an important clue to the fundamental aetiology of diabetes and which may allow the detection of the prediabetic subject, is not even mentioned. Let us hope that at least one chapter (but preferably several) will be devoted to these and related subjects in the next edition. W.P.U.J.

BIOCHEMICAL RESPONSE TO INJURY

The Biochemical Response to Injury. A Symposium organized by the Council for International Organizations of Medical Sciences. Edited by H. B. Stoner with the assistance of C. J. Threlfall. Pp. 467 + xi. Illustrated. 57s. 6d. Oxford: Blackwell Scientific Publications Ltd. 1960.

This book records the proceedings of an international symposium on the metabolic effects of injury. It covers a wide range of subjects discussed by leading research workers in this field and includes a comprehensive list of well-selected references.

Many aspects of the metabolic changes relating to such diverse injuries as burns, tourniquet shock, fractures, haemorrhagic shock, bacterial infection and surgical shock are admirably reviewed.

The possible role of bacterial endotoxin in the production of irreversible shock, although not new, is a revolutionary concept worthy of careful consideration.

Our ignorance in this field was aptly described by Dr. Haist, one of the participants, by comparing it to a blind man trying to describe an elephant. Nevertheless this book contains fundamental information and can be strongly recommended to all serious-minded doctors. G.M.P.

ATELEKTASE VAN DIE LONGE

Die Streifenatelektasen der Lunge. Von Priv.-Doz. Dr. Fr. Heuck. Pp. viii + 108. 64 Abbildungen, in 136 Einzeldarstellungen. Kartoniert DM 32.-. (Zwanglose Abhandlungen aus dem Gebiet der normalen und pathologischen Anatomie, herausgegeben von Prof. Dr. W. Bargmann und Prof. Dr. W. Doerr, Heft 7. Subskriptionspreis für Bezieher der Reihe DM 25.60.) Stuttgart: Georg Thieme Verlag. 1959.

Onder diskoïde- of streepatelektase van die longe word verstaan streepvormige skadus, 1 - 2 cm. lank, wat gewoonlik by die longbasisse op röntgenfoto's as 'n bykomstige bevinding gesien word. Die skadus is nie skerp begrens nie, en is tydelik. Vorige disseksies het gewys dat dit nie deur fibrose of pleuritis veroorsaak word nie.

Hierdie monogram bespreek 174 gevalle, en trag veral om die patologiese anatomie te bepaal. As ondersoekmetodes is o.a. brongografie, tomografie, kimografie, diereksperimente en intravitale mikroskopie gebruik.

Onder die bestaande opvattings oor die oorsaak van streepatelektase is daar baie uiteenlopende menings, bv. brongusobstruksie deur slymverstopping, verminderde beweeglikheid van die diafragma, reflekskramp van onwillekeurige spiervesels in die kleiner brongusse, ens.

Wat die lokalisasie betref, onderskei die skrywer twee tipes, nl. die intrapulmonale segmentele (of liewer subsegmentele) soort, en die subpleurale, perifêr-geleë tipe. Hy het bewys dat die brongusvertakkings in die letsels oop is, m.a.w. 'n brongus-

obstruksie kan uitgesluit word as 'n oorsaak. 'n Reflekssaamtrekking van spiervesels kon ook nie eksperimenteel bewys word nie. Die eksperimente het wel bewys dat die luginhoud van die alveoli omgekeerd eweredig is tot die bloedvulling van die kapillêre vate, en dat oorvulling van die kapillêre vate tot sigbare streepatelektase kan lei. Met geforseerde inaseming het dit weer vinnig verdwyn. Bogenoemde skyn dus die vernaamste patologies-anatomiese verandering te wees. Stimulasie van die longoppervlak het soortgelyke veranderinge gewys.

'n Mens kan net byvoeg dat die term ‚streepatelektase' dus nie die ware toedrag van sake weerspieël nie. Ongelukkig word die verhouding van hierdie toestand tot A- en B-lyne nie duidelik bespreek nie. Laasgenoemde benamings het ons nie teengekom in hierdie verhandeling nie, en ook nie die naam van Kerley nie. Daar word egter kortliks verwys na 'n ander groep ondersoekers se artikel oor pulmonele hemosiderose en stuwing. Hoewel dit 'n deeglike ondersoek is, laat dit bogenoemde vraag onbeantwoord. A.D.K.

BIOLOGY OF SEX DIFFERENTIATION

Sex Differentiation and Development. Memoirs of the Society for Endocrinology No. 7. Edited by C. R. Austin. Proceedings of a Symposium held at the Royal Society of Medicine, London, on 10 and 11 April 1958. Pp. x + 198. Illustrated. London: Cambridge University Press. 1960.

The report of this symposium — held nearly 3 years ago — contains a mass of fascinating information. Crustaceans, fish, amphibians, reptiles, birds and mammals — even insects and bacteria — form the subjects of these papers. Those of us who are restricted to the study of humans must be intrigued by accounts of hermaphroditism and sex reversal in prawns, or of sex determination and caste differentiation in the honeybee. The papers by Profs. A. Jost and S. Zuckerman are outstanding, and there is a group contribution speculating on the possibility of controlling the sex ratio at conception. At the rate at which information is being gathered one can anticipate the possibility not only of foretelling the sex of the unborn child but, perhaps, of pre-ordaining it.

In the second half of the meeting the contributions covered aberrations of human sexual development, and these are the papers that will be of most value to the clinician. Already — as a result of the recently described technique of direct chromosomal observation — many of the theories proposed have been invalidated, but this does not detract from their interest.

This volume is strongly recommended to zoologists, embryologists, physiologists, endocrinologists — in fact to all those interested in the scientific side of sex. R.H.

BIOLOGICAL SCIENCES AND DERMATOLOGY

Progress in the Biological Sciences in Relation to Dermatology. Edited by Arthur Rook, M.D., M.R.C.P. Pp. xv+480. Illustrated. 84s. net. London: Cambridge University Press. 1960.

This symposium edited by Dr. Arthur Rook is not everybody's meat.

One certain factor emerging from the study of this book cannot be emphasized enough, viz. that dermatology must be regarded as an integral part of general medicine; the remarkable advances that have occurred in the basic biological sciences have exercised their influence on specialized subjects such as dermatology as well.

There are 43 contributors. The majority are anatomists, physiologists, zoologists, biochemists, pathologists, and only 3 are dermatologists. It is difficult to review a book of this nature in detail, and I can only mention some of the important features as they strike me.

A paper by J. D. Boyd, an anatomist, deals with melanin-producing cells in vertebrates, and goes on to describe very fully and adequately the distribution of melanin pigment in man. The next 44 pages are devoted to melanogenesis and its chemistry.

To dermatologists who are interested in histochemistry the paper by A. G. Everson Pearse, particularly the part dealing with origins and early history, makes most interesting reading.

The importance of the psychosomatic concept in dermatology is fully stressed and the subsequent discussions worth reading.

I strongly recommend this edited symposium, particularly to trainee dermatologists, for the study of the anatomy and physiology of the skin. R.L.

REHABILITATION OF THE HAND

Rehabilitation of the Hand. By C. B. Wynn Parry, *M.B.E.*, M.A., D.M., D.Phys.Med., assisted by N. R. Smythe, M.A.O.T. and L. E. Baker, M.C.S.P. Pp. vii + 273 + (17). 101 figures. 51s. 9d. + 2s. postage. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1958.

During the past decade many 'hand units' have been established in the United States, Great Britain, and Scandinavia. The desirability of these specialized units is readily recognized by orthopaedic surgeons interested in the surgery of the hand. Conversely, the lack of such units is keenly felt. It is not surprising, therefore, that many books have been written on the surgery of the hand. This volume, however, fills a much-needed gap, for the aftercare of lesions of the hand is no less important than the operative technique. Adequate and well-planned pre- and postoperative care is a *sine qua non*.

This book concerns itself with detailed conservative treatment and the niceties of aftercare. There are excellent chapters on the functional anatomy of the hand and the use of electrodiagnosis. The techniques of treatment described include the finer details of physiotherapy, occupational therapy, adaptation of tools, and serial plaster stretching.

A chapter by Donal Brooks on 'reconstruction of the injured hand', and one by D. A. Brewerton on 'the rheumatoid hand and its management' heighten the interest of the book and maintain the excellence and clarity of presentation.

Those who seek the ideal will find the book an inspiration and a great help in grappling with the problems of the management of the hand affected by injury and disease. M.S.

CORRESPONDENCE : BRIEWERUBRIEK

PROFESSIONAL PROVIDENT SOCIETY

To the Editor: The gist of the plea contained in my letter[1] published in the *Journal* of 26 November was that the Professional Provident Society should remove the restrictive clause which does not allow a doctor of 51 or over to join, and also demands that he must be in good health.

Dr. E. Baskind, the Chairman of the Professional Provident Society of South Africa, in his reply[2] devotes himself mainly to a reiteration of the benefits of the society. This was not my question at all.

Dr. Baskind's specific reply to my plea is presented in one sentence — that it would be an 'uneconomic proposition'. However, he does not give any actuarial figures in support of his outright rejection.

Has he investigated how many doctors over 51 would like to join the Society? Has he computed what effect their additional claims would have on the economy of the Society? i.e. what percentage claim repayments they would constitute?

Not only does Dr. Baskind reject my plea without any supporting figures, but he states: 'Many members ... had the foresight to join when they were still healthy and under 51'. This is a statement one expects only from a commercial insurance company and not a professional provident fund.[2]

I feel that the restriction is so hard that the profession in South Africa should seriously consider whether the Professional Provident Society is not a failure, and whether a new scheme should not be inaugurated.

I ask my colleagues to submit their views.

Johannesburg *Help-hand*
13 January 1961

1. Correspondence (1960): S. Afr. Med. J., **34**, 1021.
2. *Idem* (1961): *Ibid.*, **35**, 19.